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A Monthly Review of Veterinary Science.

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FREDERICK HOBDAY, F.R.C.V.S., F.R.S.E.,
FORMERLY PROFESSOR IN THE ROYAL VETERINARY COLLEGE, LONDON.

GEO. H. WOOLDRIDGE, F.R.C.V.S.,
MEMBER OF THE ROYAL IRISH ACADEMY,
PROFESSOR OF HYGIENE, MATERIA MEDICA AND THERAPEUTICS IN THE ROYAL VETERINARY COLLEGE, LONDON.

Editor for Australasia:—

J. A. GILRUTH, D.V.Sc., M.R.C.V.S., F.R.S.E.,
DEAN OF THE VETERINARY FACULTY, UNIVERSITY OF MELBOURNE, AUSTRALIA.

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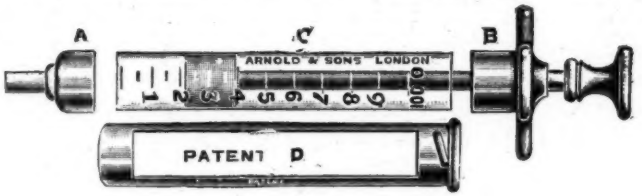

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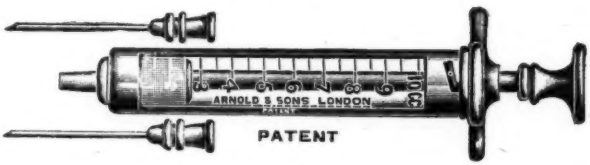

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THE

VETERINARY JOURNAL

SEPTEMBER, 1910.

Editorials.

CONTAGIOUS GRANULAR VAGINITIS IN CATTLE.

DURING the present year we have reproduced several very instructive papers in the VETERINARY JOURNAL on the above subject from the pens of such well-known Continental observers as Professor Hess and Mr. Albrechtsen, and Mr. Ainsworth Wilson in this country; and as the matter is of such vital importance to agriculturists we make no apology for again referring to it. In an editiorial note in our March issue, before Mr. Wilson's paper appeared, we referred to the fact that the disease was undoubtedly preseut in Britain, although it had largely escaped observation, probably owing to its running a somewhat benign course, and not giving rise to acute or distressing symptoms. The disease is undoubtedly responsiblein Europe and America for a considerable amount of sterility, and although the form prevailing here would appear to be much less virulent, it would be unwise to close one's eyes to the probability of its being also closely associated with sterility in this country, With this in mind, we wrote in March last: "The subject of sterility is very intimately associated with abortion, and it would confer a great boon upon the agricultural community if the commission now investigating abortion were to be instructed to extend their researches fo include sterility in cattle." Hence it is particularly gratifying to us to see in the report of the Chief Veterinary Officer of the Board of Agriculture a portion of which we reproduce on pp. 523-6 that the affection has not in the least been

overlooked. In fact, Mr. Stockman calls attention to it "on account of its very contagious nature and the possible importance of its results." We do not believe, however, that contagious vaginitis in this country is generally regarded as a cause of abortion, but that it is frequently a cause of sterility by interfering in some manner with conception.

Undoubtedly our knowledge of these contagious diseases of the genital organs is very meagre ; there is much confusion. In his paper Mr. Ainsworth Wilson attempted to differentiate between several of them, but apparently Mr. Stockman is of opinion that what the former regarded as two diseases is only one disease. In describing an acute form of "Contagious Granular Vaginitis" Mr. Stockman says : "This form is probably what has for many years been known to farmers as "bull burn." Mr. Wilson regards the latter affection as an entirely different disease—viz., vesicular vaginitis. Judging from his report and from his statements when opening the discussion on the subject at the meeting of the National Veterinary Association, we gather that Mr. Stockman doubts the existence of two forms of contagious vaginitis in cattle, one granular and one vesicular, while Mr. Wilson affirms their dual existence.

It would not be so very difficult to follow if the differences of opinion ended there, but worse is to follow. During the discussion at the National, Sir John M'Fadyean stated in no unmeasured terms that he emphatically disagreed with Mr. Wilson's statements as to the existence of contagious granular vaginitis over any widespread area in this country, a view which is obviously also in disagreement with the statement of the Chief Veterinary Officer of the Board of Agriculture. He did not, however, throw any light on the nature of the contagious disease of the external genitals of cows and bulls which undoubtedly does exist in the British Isles, and which is commonly known as "bull burn."

Amongst these variations of opinion what is one to believe ? Our view is that there are two contagious forms of vaginitis. We have frequently met in various parts of the country with a contagious vesicular vaginitis easily and readily transmissible from cows to bulls and *vice versa*. The symptoms are quite distinct from those of contagious granular vaginitis described by

Mr. Wilson, and by Mr. Stockman as a chronic form, and by the various Continental authorities.

We sincerely endorse Mr. Stockman's hope that it will be possible to conduct further inquiry into this disease, and suggest that the most desirable procedure would be to extend the scope of inquiry of the Committee on Epizootic Abortion. That committee must have obtained an enormous amount of information and material such as would be of the greatest possible assistance in an investigation into other contagious affections of the generative organs of cattle. It would be a pity, if not a disaster, to lose the opportunity, and we trust the Board of Agriculture will again rise to the occasion.

MONTHLY MAGAZINES AND THE POST OFFICE.

THE Editor of the *Agricultural Economist and Horticultural Review* is to be congratulated on the effort he is making to induce the Postal authorities in this country to modify the harassing and archaic regulations which hamper the development of the periodical press other than weeklies and dailies. It is obviously absurd that a certain number of weeklies, in many instances weighing several pounds, can be conveyed by post for a ½d., whilst scientific magazines like ours one quarter of the weight have to pay 1½d., per issue. In no respect are we further behind the United States in commercial and agricultural enterprise than here. Our contemporary points out, for instance, that five copies of that journal, if published in the United States of America, could be sent through the post to any village or hamlet in that vast territory for 1d.; while in Great Britain every single copy posted costs 2d. The Post Office here absorbs nearly 40 per cent. of the subscription revenue of the paper; in the United States it would only claim a moderate 5 per cent., or less. This tax cripples the development of the scientific and lay press in this country. We cordially support the effort now being made to convince the Post Office authorities that the time has now come to make a drastic modification of the out-of-date hindrances to the development of magazine literature.

General Articles.

SOME INJURIES TO THE FLEXOR TENDONS AND SUSPENSORY LIGAMENT.[1]

By J. MACQUEEN, F.R.C.V.S.

Professor of Surgery in the Royal Veterinary College, London.

In discussing this subject it is desirable briefly to recall the arrangement and relations of the structures exposed to injury.

Fore-limb.—The flexor perforans muscle, three times larger than the perforatus, arises from the humerus, radius, and ulna, and extends to near the knee where its tendon begins. The tendon passes through the carpal arch to the middle of the canon, where it is joined by the sub-carpal ligament, then downwards to the fetlock, where it passes through the perforatus ring and over the sesamoid pulley. Descending behind the pastern, under the bifid insertion of the perforatus and over the glenoid prominence and navicular bone, it is inserted on the semi-lunar crest of the os pedis. Slightly compressed at the knee, it is rounder at the canon, considerably expanded and flattened at the fetlock, narrowed though still flat behind the suffraginis, at the os coronæ, to which it is loosely attached, it again expands and rapidly attains its greatest breadth at its insertion. In volume it varies little from its origin to the point of junction with the subcarpal ligament, and below this the increase is hardly noticeable owing to the gradual attenuation of the reinforcing band. At the fetlock the tendon is thicker, and its anterior surface, moulded on the sesamoid pulley, shows some of the characters of fibro-cartilage. Another increase in thickness and firmness occurs at the os coronæ. It appears to be weakest at its terminal expansion which, however, is well supported by the posterior digital ligament.

The sub-carpal or "check" ligament, a direct continuation of the posterior common ligament of the knee, is united at its origin to the anterior fibrous wall of the carpal arch and the suspensory ligament. Descending, it closely embraces the anterior surface of the perforans, which it appears to join at the middle of the canon. In many instances their fushion is very gradually effected, as some indication of the parts of tendon and ligament can be traced to near the fetlock. The sub-carpal ligament is the strongest portion of the suspensory apparatus of the fetlock.

The perforatus or superficial flexor muscle, arising with a portion

[1] Portion of paper read at the National Veterinary Association Meeting at Harrogate.

of the perforans from the humerus, extends to near the knee where it is succeeded by tendon. In close contact throughout with the perforans, the tendo-perforatus passes through the carpal arch to near the fetlock, where it forms a sheathlike ring for the perforans, then descending and becoming somewhat broader it terminates by a bifid insertion on the os coronæ. Before entering the carpal arch the perforatus is joined by the radial ligament, which hitherto has attracted little attention outside the dissecting room.

The radial ligament arises from the inner border of the posterior surface of the lower extremity of the radius and extends obliquely downward and outward to join the perforatus tendon. It is a short, rather lax fibrous band, between 2 and 3 in. long, about an inch broad, and less than ½-in. thick. Recently in Germany and France, cases of lameness have been attributed to strain of this ligament.

The suspensory ligament, arising from the lower row of carpal bones and the head of the canon, descends between the sub-carpal ligament and metacarpus to near the "buttons" where it bifurcates. Each branch is implanted on the excentric surface of the corresponding sesamoid, and a portion of each band is continued downward and forward to join the extensor pedis tendon. From its origin to the point of bifurcation the suspensory is flattened and closely applied to the canon, its branches to the sesamoids are rounded, and the extensor bands are flat. It has a covering of connective tissue which attaches it to the canon and flexor aponeurosis. In structure it differs from the tendons by containing fasciculi of striped muscle and some fat.

Hind-limb.—Apart from their points of origin and a few other differences, the more important features are: The perforans in the tarsal arch is not accompanied by the perforatus; at the upper metatarsal region it is joined by the tendon of the accessory flexor muscle, and near the middle of the shank by the sub-tarsal ligament, which though longer is less thick or strong than the sub-carpal ligament. The perforatus has a very short muscular portion, and its tendon, beginning just below the upper third of the tibia, after a winding course, reaches the point of the hock, where it forms a cap for the summit of the os calcis. Below the hock the perforatus descends the shank, as in the fore limb, to the os coronæ.

Tendon is made up of groups of parallel white fibrils, interspersed with flattened nucleated connective tissue cells, arranged in rows running in the direction of the tendon fibres. Between the bundles are inter-fascicular spaces, and primary and secondary connective tissue septa, continuous with the peritendinous covering. The nerves

(few and non-medullated), blood-vessels, and lymphatics ramify in the septa. The sub-carpal and sub-tarsal ligaments have thicker interfascicular septa and are more vascular than the tendons.

Peritendineum.—Each tendon has its own covering of connective tissue. This is composed of several laminæ, more or less united, closely investing the tendon, continuous inwardly with the interfascicular septa, and connected outwardly, according to the part examined, with the visceral layer of the flexor synovial sheath, the common aponeurosis, or the adjoining tendon or ligament. Between its laminæ the vessels and nerves break up to penetrate the interfascicular septa of the tendon.

Aponeurosis.—The metacarpo-phalangean or common aponeurosis furnishes a subcutaneous covering to the flexor tendons and sub-carpal ligament and separates these from the suspensory. It consists of two principal layers of fibrous tissue, united to each other and to the tendons or parietal synovial sheath by areolar tissue and continuous with the posterior wall of the carpal arch and fascia of the forearm. It forms a strong fibrous brace for the flexor tendons at the sesamoids and pastern, and supports and protects the vessels and nerves. In the hind limb the metatarso-phalangean aponeurosis is similarly arranged.

Synovial sheaths facilitate movement of the tendons, consisting of two parts continuous with each other; a parietal, lining the aponeurosis or other supporting tissue, and a visceral, investing the proper covering of the tendon. The opposed surfaces are lined with endothelium.

The carpo-metacarpal sheath, extending from about 2 inches above carpus to near the middle of the canon, lines the carpal arch and part of the metacarpal aponeurosis; reflected on both tendons in the carpal arch and below on the perforans, posterior surface of the sub-carpal ligament and anterior surface of the perforatus tendon. From an inch or two below the carpal arch to the upper margin of the sesamoid sheath the perforatus has no synovial membrane on its posterior surface, and the anterior surface of this portion of the tendon is separated by loose connective tissue from parietal synovial membrane of the perforans.

The great sesamoid sheath extends from the level of the "buttons" to the middle of the os coronæ, where it is separated from the navicular bursa by an attachment of the perforans, lining the aponeurosis and sesamoid pulley and reflected on flexor tendons, being modified at the sesamoids by the absence of endothelium on the anterior surface

of the perforans. There is no synovial covering on a portion of the posterior surface of the perforatus, which is united to the aponeurosis of the fetlock.

The navicular bursa or sheath extends from the middle of the os coronæ to below the navicular bone, and forms a short synovial sac between the perforans and the navicular bone and its interosseous ligament.

Strain.

Strain, partial rupture of tendon or tendinitis, varies in gravity with the degree of injury, the position of the strain, and the cord affected. Strain may be slight and limited to a few axial or peripheral fibres (tendinitis), or severe, and involve a considerable portion of the thickness of the tendon, including its connective tissue covering (paratendinitis). In strain affecting few or many fibres the ruptures may occur at one level (short strain), or owing to subsequent tearing or secondary ruptures at different levels, the laceration may represent a sloping breach of the tendon (long strain). In most cases there is a centre of limited rupture, but owing to consecutive inflammatory changes the distension extends above and below the seat of injury. In this way, amongst others, the diffused thickening of the sub-carpal ligament and the elongation of the primary distension of "bowed" perforatus are produced.

Position of Strain.—The perforans, though far less frequently affected than is generally supposed, may be strained just under the knee, at its junction with the sub-carpal ligament, at the sesamoids, behind the pastern or in the "heel pan," where it is very difficult to distinguish distension of this tendon from rupture of the digital aponeurosis or bursitis of the synovial sheath. The sub-carpal ligament may be strained close to its fusion with the perforans, near its origin below the knee, or towards the fetlock at its real termination. The strain may be short or limited to an inch or two of the ligament, but as there is a constant tendency to extension of the inflammation, most often the distension is diffused.

The perforatus may be strained at the knee, at the middle of the canon, or at the fetlock. Occasionally strain occurs at other points, as within the carpal arch and at the bifurcation of the tendon behind the pastern. Strain of the radial ligament as a primary injury must be very rare, though acute bursitis of the carpal sheath, caused by bruising, sometimes extends to this ligament.

Strain of the suspensory usually occurs immediately above its

bifurcation or affects the inner branch. In dilaceration or upward splitting of the suspensory the apparent strain may extend to the upper third of the ligament.

The relative frequency of strain at these sites cannot be possibly stated, because cases are seldom seen early enough to permit of accurate diagnosis. But perhaps the commonest strain of the perforatus is represented by "bowed tendon" at the middle of the canon, that of the perforans at the fetlock, and that of the subcarpal ligament near its junction with the perforans.

Regarding the comparative frequency of perforans, perforatus, sub-carpal, and suspensory strain there is hardly more justification for confident assertion. However, one may say that in cart horses the sub-carpal ligament is more often strained than the perforans, that by extension of inflammation the perforatus is often implicated, and that strain of the suspensory is very seldom seen. In saddle horses, racehorses, chasers, hunters, and polo ponies strain of the perforatus or of the suspensory is more common than strain of the perforans. Anterior strains are much more frequent than posterior strains.

Statistics and published cases of flexor strain should not be accepted without caution. Repeated examination of the leg is required to distinguish the tendon strained, and in many cases some doubt remains. Poy, analysing 230 cases of anterior strain in horses engaged at fast work, gives 116 suspensory alone, 17 suspensory and one tendon, 34 perforatus, 30 both flexors, 16 sub-carpal ligament, 10 sub-carpal and one tendon, and 7 perforans. Barrier found the suspensory affected five times in eleven cases, and Jacoulet thirteen times in eighteen cases of strain in saddle horses; and in six months at Saumur, Joly had 75 cases in horses particularly exposed to strain by attenuation of shock: 25 perforatus, 17 suspensory, 17 affecting the tissues connecting the perforatus and sub-carpal, 1 perforans, and 8 affecting the tendons without possible distinction. Lesbre, from observations made in the dissecting-room, considers that the sub-carpal ligament and perforans are more frequently strained than the suspensory. Other observers have found the suspensory affected 24, 50, and 54 times in 100 cases of strain, and Pader states that probably many cases of apparent distension of the suspensory may be due to *Filaria reticulata*, as in 37 cases of parasitic invasion of this ligament, 13 presented enlargements identical with the lesions of chronic strain. In nine years, according to Fröhner, 36,230 horses of the Prussian Army were treated for inflammation of the flexors and tendon sheaths, and of this number 70 per cent. were affected in both

flexors of one limb, and 20 per cent. in the suspensory. The right fore leg was affected in 43 per cent., and the left in 36 per cent. of the cases. Later German statistics give, for one year, 2,695 cases: 1,090 both flexors, 495 suspensory alone, 401 perforans alone, 286 perforatus alone, 120 both flexors and suspensory, 49 sub-carpal ligament, 1 radial ligament, and 45 synovitis or rheumatic tendinitis. In the French army the admissions for strained tendons and fetlocks were 1,080 in 1891, and 3,923 in 1897. In the British Army, with an effective home strength of 21,000 horses in 1907, the admissions for strained tendons were 634, and for strained ligaments 834. Unfortunately the distribution of these strains is not given in the report from which these figures were obtained, but the small number of cases is both remarkable and gratifying.

Causes.—Predisposing, or contributing and exciting causes, are recognised, but as many of these appear to be interdependent they will be placed together. Defective conformation is represented by long, upright, or too oblique pasterns, buck knees, crooked legs, and tied-in tendons. Bent knees, especially when congenital, should be excepted, as they appear to save the tendons, and in this belief some owners and trainers prefer horses that lean a little at the knees, Tendons, though thick and apparently strong, may be weak, defective in quality, of unequal density, less resistant, and more liable to strain; these defects being due to heredity, breeding, rearing, or dieting. Other causes are premature work or training before complete growth or consolidation of the tendons and ligaments; forced training or pushing the preparation of the young horse; nervous excitability in thoroughbred and some other horses, by inducing more energetic efforts; fast work, especially racing, chasing, hunting, and trotting (perforatus and suspensory strains); muscular fatigue towards the finish of a fast run race or in galloping too far, and weak flabby muscles, by relaxing the flexors and too suddenly throwing the support of the fetlock on the tendons; heavy draught work, starting or backing a load, holding back going down hill, and shunting (sub-carpal or perforans strain); antecedent disease, adhesions, with or without shortening, worn tendons, chronic disease of tendon, aponeurosis, or synovial sheath: hard ground, by increasing the flexion of the pastern and the tension of suspensory and perforatus; heavy holding ground by detaining the foot, impeding movement and increasing effort, ground with a soft or movable surface-layer as grass after rain, and wood or asphalt when wet or greasy, by causing slipping; ringbone and enlargements of the pastern by hindering or preventing

movement of the joints and consequent relaxation of the tendons (perforans strain); long toes and low heels and shoeing with tips predispose to strain of perforans and perforatus. Slipping in the stable or on the road, a false step, and in a tired horse a sudden acceleration of speed may cause strain.

In forming an estimate of the merits of these causes attention should be given to the action of the fetlock and pastern. The fetlock, with its angle open in front to about 150°, is sustained by the suspensory and supported by the flexor tendons. The suspensory yields a little when the fetlock sinks and regains its former length when the fetlock rises. In movement, the tension of the suspensory is in proportion to the degree of dorsal flexion of the fetlock, and if it is elastic the property of stretching is due to the muscular fasciculi incorporated with its fibrous tissue. Immediate *post-mortem* examination of the suspensory reveals no sign of special elasticity, and in the thoroughbred during movement it appears to be rather thicker when relaxed. Pader holds that the suspensory is not more elastic than the tendons, and Lesbre suggests that the lengthening or yielding of this ligament is more apparent than real because of its bifurcation and the position of its attachments to the pastern. Implanted on the free or outer side of the sesamoids, it acts like a cord suspending the pulley by its axis, and when the fetlock descends the sesamoid mass passes between the branches of the ligament. However this may be, the suspensory acts by a direct pull on the sesamoids, while the flexor tendons furnish a movable support to the fetlock, and together tendons and suspensory may be regarded as a single power acting at the sesamoids on the lever of the pastern to oppose the descent of the fetlock and closure of its angle in front. In the standing position the weight transmitted through the canon falls on the fetlock where it meets the inclined articular surface of the pastern and is resolved into two parts—one perpendicular to the inclined surface, the other parallel to it. The perpendicular weight exerts its pressure on the pastern bones which neutralize it by their resistance, while the other part, which abuts behind on the sesamoids, tends to force the fetlock downwards by inducing oscillation of the pastern lever with consecutive closing of the metacarpo-phalangean angle. This tendency is overcome by the tension of the suspensory apparatus, which keeps the fetlock in position and preserves its normal angle. In locomotion, when the foot comes to the ground, the pressure supported by the fetlock is represented by the weight of the body plus the force of inertia resulting from the acquired speed. The more rapid the pace the greater

the pressure; and while the foot is in contact with the ground the fetlock, at first, is a centre of attenuation of shock and afterwards a centre of impulsion. Its mechanism was investigated in 1891 by by Barrier and Siedamgrotzky. Working independently, with the assistance of instantaneous photography, they arrived at very similar conclusions, which as regards the causation of strain may be shortly stated: When the foot is coming to the ground the oblique canon forms with the pastern a straight line (fig. *a*, 2). When the foot is in support the canon becomes vertical, while the pastern approaches more or less the horizontal. In virtue of the marked dorsal flexion of the fetlock, the pastern or phalangean lever oscillates in two different directions; forwards and upwards on the canon and downwards on the os pedis and navicular. These two movements of oscillation are absolutely simultaneous, and they produce at once closing of the angle of the fetlock and flexion of the coffin joint (fig. *a*, 3). But the effect of this closing of angles is different on each flexor tendon. The perforans representing a cord extending from the head of the canon (origin of sub-carpal ligament) to the os pedis is relaxed, the inflexion of the pastern having shortened the distance from its two points of insertion. The perforatus, extending ftom the radial ligament to the base of the pastern is made tense, in consequence of the separation of its points of attachment, and for the same reason it more closely braces the fetlock. Towards the middle of this stage the suspensory reaches its maximum of tension and is most exposed to rupture.

From this moment of the support the angle of the fetlock effects a sort of forward oscillation, the pastern straightens, and the flexion of the coffin joint progressively diminishes, whilst the canon is inclined forwards (fig. *a*, 4). This oscillation of the fetlock and the simultaneous straightening of the pastern have the effect in a fast pace—as a striding gallop—of effacing the angle which was formed at the hoof, and of producing a little greater closing of the metacarpo-phalangean angle. Under the influence of these two causes the tendons are immediately stretched and quickly reach the limit of their extension. The more extensive the oscillation of the angle of the fetlock on the articulation of the foot the greater the tension of the sub-carpal ligament, so that at this instant the least supplementary effort may cause a rupture.

Inferentially these observations may be summarized: (*a*) strain of the suspensory is produced by excessive tension (paratonia) of the ligament at the moment of greatest dorsal flexion of the fetlock, when

by inflexion of the coffin joint the semi-lunar crest is brought nearer the sesamoid pulley and the perforans is relaxed (fig. *a*, 3); (*b*) the perforatus may be strained in the same way as the suspensory, with which it shares the charge of supporting the fetlock when the foot comes to the ground; (*c*) strain of the sub-carpal ligament or perforans is produced by excessive tension (paratonia) of the tendon at the moment

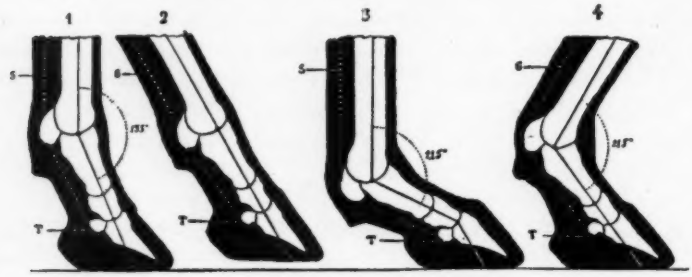

FIG. A.

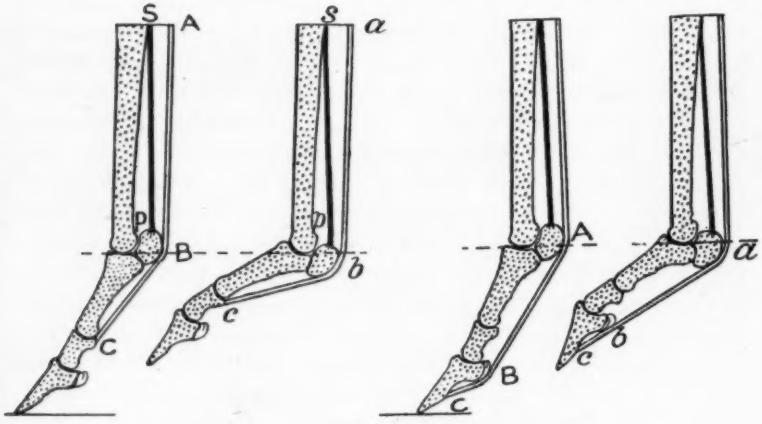

FIG. B.

of hyper-extension of the pastern during impulsion in fast work or in forced draught (fig. *a*, 4).

In the first stage of the support (fig. *a*, 3) some hesitation arises regarding the action of the perforans and sub-carpal and their escape from strain. The very limited relaxation of the perforans tendon at the coffin joint can hardly be sufficient to permit the angle of the fetlock to close to the extent seen in racing, when the ergot almost

touches the ground. The tendon is inextensible, and its apparent elongation when the fetlock descends should be conferred to the *muscle*. The sub-carpal ligament is not constantly tense, and it is reasonable to suppose that the perforans muscle is stretched before this ligament is called upon to share the pull on the tendon. In racing and similar fast paces muscles sooner or later become fatigued, and as they are not continuously contracted, when strain of the sub-carpal does happen at this stage, most likely the injury occurs during relaxation of the muscle, when the ligament, unassisted by muscular contraction, is in extreme tension.

Regarding strain of the perforatus the operation of the cause is not clear. At the fetlock this tendon moves freely on the perforans, and, according to Joly, with the advantage of having watched the action of the naked flexors in a living horse, the perforatus begins to move and comes to rest a little sooner than the perforans, which appears to continue the movement. The perforatus muscle is small, presumably weak and easily stretched, and the radial or check ligament is lax enough to allow for elongation under tension. No doubt partial rupture of this tendon sometimes occurs, but clinical observation suggests that the usual distension—"bowed tendon"—is gradually produced by repeated limited laceration of the peri-tendinous covering, and that later the body of the tendon becomes affected by extension from the peritendineum (paratendinitis).

Effects of Strain.—Coleman taught that sprain is "an inflammation of the cellular tissue connecting the perforatus and perforans together"; and Percivall, while holding that tendons are incapable of extension, and are too firm and strong to sustain hurt from common accident, recognized the fact that they are surrounded by a soft, delicate tissue, which must, every time they are forcibly pulled or stretched, be extremely liable to laceration. Since then—excepting those who perhaps too seriously interpreting the words of the preacher, "he that increaseth knowledge, increaseth sorrow," have been content with offering opinions—very few have taken the trouble to inquire into the nature of strain, and Pader, Veterinary-Major in the French army, has done more than any other to explain the pathology of tendinitis.

Excessive tension of a tendon causes rupture with retraction of fasciculi, laceration of the peritendinous covering and interfascicular septa, extravasation, and sero-sanguineous exudation. The exudate fills the interfascicular spaces and distends the areolar septa, the hitherto quiescent tendon cells increase in size and become active,

while the damaged fibres are partially converted into a structureless hyaline pulp. Inflammatory reaction is set up, and in the reparative process the breach in the tendon is made good by granulation tissue, which is formed by the agency of the tendon cells and the fibroblasts of the connective tissue adjacent to the lesion. Eventually the new-formed tissue undergoes cicatricial contraction. Though tendinitis, caused by strain, is an aseptic process it nearly always produces permanent distension at the seat of injury. Resolution is a possible termination, but in most cases of sufficient intensity to attract attention, repair is imperfect, and after apparent recovery it is exceptional to find the tendon normal or quite free from adventitious growth. As a rule there remains a nodular or diffused thickening of the tendon or peritendinous covering. A tendon once inflamed is predisposed to relapse, and too frequently, owing to insufficient rest, to position, or to persistent irritation the result is chronic tendinitis.

Post-mortem examination of a chronic or indurated strain of the sub-carpal ligament shows, in addition to remarkable intrinsic distension of the ligament, considerable peripheral increase from new formation of connective tissue. The suspensory lying in front is not involved, but the borders of the sub-carpal ligament are firmly united to the perforatus, and its covering by the enormously thickened aponeurosis. This union forms a tube for the perforans, which remains free with the synovial sheath. At the point where the sub-carpal joins this tendon, and below to a variable extent, the perforans is more or less distended, but above within the carpal sheath the tendon may be normal, atrophied, or hypertrophied, according to the intensity and duration of the inflammation, and the degree of compression exerted by the surrounding new fibrous growth.

Partial atrophy of the perforans may result from chronic inflammation of either the sub-carpal ligament or perforatus. Hypertrophy may be due to a compensatory cause, the perforans, owing to failure of the perforatus or one of the ligaments, being more actively engaged in supporting weight. But sometimes the increase in thickness is pathological and due to tendinitis, either primary or arising by extension from the sub-carpal ligament, perforatus, or aponeurosis. The metacarpal vessels and nerves running through the thick fibrous growth under stress of increasing compression may be injured, and in fact the wall of the metacarpal artery is much thickened, and the nerves, including the branch which crosses the perforatus, are expanded and flattened, but otherwise they appear normal. Sclerosis, arteritis, and phlebitis of the vessels, and sclerosis

and perifascicular exudation of the nerves, have been observed by Pader, and he suggests that possibly this interstitial neuritis may contribute to the lameness.

Similar peritendinous alterations with ultimate ossification of the fibrous growth occur in connection with chronic tendinitis of the perforans at the fetlock, usually of the hind limb. The rupture occurs between the sesamoids and the os coronæ, where the perforans is constricted and relatively weak. The tendon presents a diffused distension extending above the sesamoids, and forming a notable prominence behind the pastern. The synovial sheath and aponeurosis, inflamed and thickened, appear more seriously affected than the tendon. Some observers, probably impressed by the more obvious lesion, maintain that synovitis precedes the tendinitis; but Siedamgrotzky, after numerous investigations, held that in every case of chronic inflammation of the sesamoid sheath accompanied by lameness and tumefaction, the perforans has been partially ruptured, and that the synovitis is the result of extension from the tendon. *Post-mortem* examination of a chronic case does not reveal the order of attack. Both tendons and the synovial sheath may be affected, the perforans by distension, the perforatus by peritendinous increase, and sometimes the aponeurosis shows more extensive alteration than either the tendons or synovial sheath. In the fore-limb, at the perforatus ring, the perforans alone may be distended coincidently with inflammation of the sesamoid sheath. In this case the synovitis appears to precede the tendinitis, which invades the sesamoid surface of the tendon.

Tendinitis of the perforatus at the middle of the canon usually arises from a very limited initial injury of the tendon or the peritendinous covering. At first the leg is merely filled or œdematous over a portion of the tendon, and the lameness, not very marked, disappears with the swelling under rest and bandaging. Gradually, however, the tendinitis extends, the swelling becomes firmer and persists in spite of bandages and simple remedies. At this stage careful treatment may arrest the progress of the inflammation, but in most cases insufficiently rested the peritendinitis invades the aponeurosis, the borders of the perforatus become united to the subcarpal ligament, and permanent distension and adhesions extend far beyond the seat of primary injury.

In high strain and in strain at the fetlock the distension of the perforatus is complicated by synovitis of the carpal or sesamoid sheath, which always aggravates the lameness and retards recovery.

Here mention may be made of those cases of "diffused thickening of the flexor tendons" of one or both fore- or hind-limbs. The leg from knee or hock to fetlock is enlarged over the tendons by persistent, firm, not very sensitive swelling, without abrasion or lameness, and causing no inconvenience beyond raising the heels slightly off the ground. In this condition the tendons are not really thickened, but the aponeurosis and peritendinous coverings form a dense fibrous shield which appears to interfere very little with the movement of the flexors. What is the cause? Wear and tear, constitutional weakness, heredity, or what? Though sometimes referred to rheumatism, the alteration gradually produced is so painless in its progress and effects that one hesitates to accept this explanation. A more feasible answer may be found in the effects of excessive work, and probably the chronic peritendinous thickening arises from the stress of constant heavy traction in cart horses, and from jumping and prolonged galloping in hunters and other saddle horses.

In strain of the suspensory above its bifurcation the distension at first is lax and compressible, but as repair advances the injured part becomes harder. Its connective tissue covering participates in the inflammatory process, but the peripheral increase is less extensive than in flexor strain. The recessed position of this portion of the ligament and its separation from the sub-carpal and tendons by the aponeurosis and a connective tissue layer possibly explain the frequent escape of the tendons from invasion. But in strain of one of its branches the consecutive inflammation not only distends the suspensory but extends through the medium of the aponeurosis to the perforatus and lateral sesamoid ligament and produces one-sided chronic enlargement of the fetlock.

Parasitic invasion of the suspensory and perforatus has been observed in Austria, Russia, and the South of France. The parasite (*Filaria reticulata*) in excavating galleries causes the formation of painless nodular enlargement of the ligament, which may be mistaken for old-standing distension.

Filariosis of the suspensory has been investigated in Russia by Tchulovski, and in France by Pader, and a full description of the condition will be found in the *Journal of Comparative Pathology* for December, 1908. In one district Pader examined *post mortem* 43 horses, asses and mules, and found 35 affected in various degrees; and Tchulovski had 51 cases in 53 horses examined at Kazan. Apparently, while active, the parasites weaken the resistance of the suspensory and predispose it to strain or rupture. After a time the

parasites perish and the lesions are repaired, but the ligament remains more or less enlarged. So far the writer has not been able to find a suspensory with the parasite. In a few foreign-bred ponies the ligament of one or both forelegs has been found nodulated as in filariasis, but the cause could not be ascertained. In this country owners are not readily persuaded to sacrifice useful animals to gratify surgical curiosity.

Symptoms and Diagnosis.—The symptoms of strain being familiar, only a few will be referred to here. Diagnosis bristles with difficulties fully appreciated by the writer, who being himself unable to see clearly, can hardly show the way to others. Every strain is accompanied by the usual signs of inflammation, and soon after the injury, swelling, pain, and increased heat of the strained part are discoverable. These symptoms, as well as lameness, vary not only with the measure of the inflammation but to some extent with the position of the strain and the tendon or ligament involved. Lameness is more marked in sub-carpal or perforans strain than in strain of the suspensory or perforatus. Severe strain of the sub-carpal or perforans appears to be much more painful than other strains of equal extent or intensity. The horse walks lame and may be unable to trot. In strain of the perforatus ("bowed tendon") or suspensory, walking, may be free from nodding, and trotting may be comparatively easy. The difference in degree of the lameness exhibited in the two cases is due partly to function, and partly to synovitis, which is almost a constant accompaniment of perforans strain, and only an occasional complication of strain of the perforatus. Excepting an inch or two at the mid-canon and at its termination, the perforans tendon throughout is invested by synovial membrane, while the perforatus is only so covered in places. The horse's action may be suggestive, but in few cases does action alone warrant the diagnosis of strained tendon. Inspection of the lame leg, except in recent bowed tendon, may mislead, and a case sometimes occurs in which the exception should be disregarded to avoid error. Painful œdema over the tendons is helpful, but its many causes unconnected with strain should be considered. Manipulation of the injured region is most useful in forming an opinion of the nature of the case, and there is no better method of arriving at a differential diagnosis of the cord affected. In practice the advantage of distinguishing the strained from the unstrained tendon or ligament may not be very great, treatment varies very little, and often the diagnosis "strained tendon" is sufficient, and perhaps it is fortunate that the horse owner in this particular is not more exacting. Differential

diagnosis of many cases is extremely difficult and of some impossible, even after repeated examination of the leg. But bearing in mind that in most of the commoner strains the inflamed peritendinous tissues establish an intimate and more or less extensive connection between the tendons, a comprehensive view may be taken by anticipating probable extension of the tendinitis to parts which are neither distinctly normal nor yet clearly affected. In this way lies safety in diagnosis. The observer may be confident and fully justified in diagnosing strain of the subcarpal ligament and at the same time quite unable positively to state whether or not the perforatus or perforans is also implicated, but his experience of similar cases should enable him to affirm that the perforatus will not escape.

In diagnosing strain just under the knee, the sub-carpal, perforans, perforatus, carpal sheath, and the aponeurosis merit consideration.

Firm painful swelling near the canon and extending 4 or 5 in. downwards usually denotes, in the carthorse, sub-carpal strain, and in others strain of the perforans with synovitis of the carpal sheath. Superficial swelling extending upwards and distending the carpal arch generally arises from perforatus strain, and when the tumefaction is one-sided and very painful external injury may be the cause. Clipping the hair over the tendons facilitates further examination. By flexing the knee and tracing singly the ligament and tendons a centre of tendinitis will be discovered. Later, in a week or two, the inflammation having extended, while a diagnosis of strain can be safely made, uncertainty remains as to the cord primarily or mainly affected. In strain of the sub-carpal ligament at its junction with the perforans, the resulting distension appears to involve the tendon as well as the ligament, but *post-mortem* examination shows, at least in a few cases, that the lesion is restricted to the portion of ligament that is joined to the tendon. In young horses lame from high splint, œdema may extend to the adjoining aponeurosis and excite suspicion of flexor strain. Doubt will be satisfied by applying a bandage, which generally removes the swelling.

Strain of the perforatus at the middle of the canon may develop insidiously, or it may be shown by immediate conspicuous engorgement over the tendon. The slowly progressive case at first may be overlooked, because there is very little interference with function, but the other at once attracts attention. The horse is very lame, and the swelling, quickly formed, may have to be reduced before the nature of the injury can be ascertained. Usually the strain is severe and accompanied by acute tendinitis, which rapidly produces fusiform

deformity of the tendon with extensive peritendinous thickening. The progressive case begins by the formation of a warm, slightly painful, compressible swelling over the tendon. This may be attributed to a blow, but there is no abrasion; though the absence of signs of contusion on a leg bandaged at work should not altogether exclude external injury as a possible cause. If arising from strain the œdema diminishes or disappears under bandages and re-forms when the leg is left uncovered. Lameness is hardly noticeable, and the horse may not be rested. In time, depending on the degree of injury and the horse's work or exercise, the swelling extends, becomes harder, and does not yield to lotions or bandaging. The tendon and its covering are inflamed, and short or elongated permanent distension will result. Owing to various causes, the horse is kept going, meanwhile peritendinitis extends, further laceration may occur, adhesions form, and eventually the condition of the leg and increasing lameness enforce rest and treatment.

Differential diagnosis of strain at the fetlock, fore or hind, is seldom easy. Occasionally, and only at the beginning, a case which appears to represent a flexor strain may be entirely due to synovitis of the sesamoid sheath, and as the perforans cannot long escape invasion, the cause of lameness may be puzzling. The condition of the inflamed synovial sheath hinders exploration of the tendons. Both flexors may convey the impression of distension, while only one is inflamed; and in the chronic case paratendinitis (aponeurosis and synovial sheath) prevents intimate examination of the parts. Sometimes the position of the fetlock or the heels assists in the diagnosis of suspected flexor strain. Synovitis with knuckling, the foot resting flat on the ground, suggests perforans strain; synovitis with raised heels points to perforatus strain, and paratendinitis or aponeurotic thickening; and synovitis with knuckling and raised heels to tendinitis of both flexors. Firm painful swelling over the flexors below the the sesamoids usually arises from perforans strain, complicated by synovitis and paratendinitis, and much of the thickening or bulging behind the pastern proceeds from changes in the aponeurosis. In some cases there is distension of the inferior sesamoid ligaments.

Recent suspensory strain may be recognized very easily, but diagnosis of the chronic case of branch strain may be rendered very difficult owing to adventitious growth and adhesions. The affected ligament should be traced in the raised limb, and compared with a healthy one. Distension caused by strain is rapidly formed and always more or less diffused. Lameness varies, and considering the

condition of the ligament, it may be little marked. After rest and treatment functional recovery is the rule.

Prognosis of Strain.—The discussion of prognosis, in the absence of the horse, is unlikely to be profitable, and this part of the subject will be passed with the remark that of the various strains, considered functionally and economically, sub-carpal is the most serious. The others, placed in the order of diminishing importance, are perforans, perforatus, suspensory. This order should not be regarded as inflexible, but merely as the outcome of experience of average cases.

Treatment.—Treatment of a recent case comprises rest, which too often is ridiculously short and inadequate, raising the heels, warm fomentations to mitigate pain, ease tension, and promote absorption; douching with cold water, hosing the leg, or applying lotions of ammonium chloride, magnesium sulphate, sodium chloride, zinc acetate, alum, or lead subacetate, to remove congestion, lower local temperature, modify the inflammation and hasten absorption of the exudate. Ice poultices and continuous irrigation with cold water have similar effects, while warm moist compresses renewed every half-hour are strongly recommended by Möller, and dry uniform compression by means of cotton wool and bandages, by Hunting. A thick layer of a mixture of hard and soft paraffin containing white lead or finely powdered Epsom salt applied to the strain, and covered with cotton wool and a bandage, is said to be beneficial. Evaporating lotions, anodyne liniments and astringent paste are also employed; but probably encasing the shank with cotton wool held securely in position by an elastic or rubber bandage, and keeping the horse as far as possible at rest, will be found equally efficacious. The early treatment of an acute case continued for from four to ten days, or longer, lessens or removes the œdema, heat and pain, and isolates the firm, more or less extensive and still sensitive distension, which is now in process of repair. For some time the lesion remains apparently stationary, and though the preliminary remedies, supplemented by hand rubbing or massage may be continued, a cold water bandage, or a compress soaked in saturated solution of Epsom salt, applied to the leg night and day, may be found sufficient to prepare the case for more active treatment.

Massage as a therapeutic measure in human and veterinary practice was introduced by Girard, Veterinary Surgeon to the Imperial Guard of France in 1857, and its rational application to strained tendons has been explained by Waldteufel. He uses an antiseptic pomade composed of lard 250 parts, cresyl 10 parts, to

which water 100 parts is gradually added and thoroughly mixed to form a cream. After smearing the swollen part with the pomade, massage proceeds by very gently rubbing, from below upwards, all round the strain to unload the vessels, and to remove extravasation. Progressive pressure with the palm or the palmar surface of the thumb and fingers should then be applied to the strained or painful parts, and always in the direction of venous return. After rubbing for fifteen minutes or so the part becomes softer and less sensitive. Then the pressure should be stronger and the movements of the hand accelerated for a further fifteen minutes. Massage should be practised for half-an-hour once or twice daily according to the case, and in the intervals the leg should be bandaged. This treatment gives excellent results in cases associated with much œdema.

Stimulation or resolvent treatment is indicated in the stationary period while repair is taking place, or in two or three weeks after the accident, but some practitioners, immediately after diagnosis, apply a mercurial all over the strained region. The favourite agent is oleate of mercury or diluted mercurial ointment.

Fröhner recommends the treatment which he saw employed at the Imperial Stables, Vienna. After the usual preliminary applications to the strained part, the hair is clipped and the skin washed and disinfected, then biniodide of mercury ointment (1 to 4 or 5) is rubbed in thoroughly for fifteen minutes, and covered with absorbent cotton and an ordinary bandage, which are allowed to remain in position for two weeks. After twenty-four hours the bandage becomes moistened with exudate, which soon dries. By resorting to this method the application of the cautery to chronic cases may not be required.

Cagny, with a large racing practice in the treatment of strain, in place of vesicants and sometimes firing, employs subcutaneous injection of a drachm of rectified oil of turpentine, to which he adds a small proportion (1 in 50) of a 5 per cent. alcoholic solution of guaiacol, to render it aseptic and to diminish the pain following the injection. This method produces much additional swelling and frequently abscess formation, which, however, is seldom serious. He claims that the results are as good as those obtained from vesicants or firing, the leg is not permanently blemished, and this remedy is more easily applied than the cautery, but he admits that some horses are much agitated for several hours after injection, a few refusing to feed for two or three days, that the œdema may be so enormous as to prevent all movement for a week, and that it is slow in disappearing.

For perforatus strain, Joly, of the Training School, Saumur, in 1901, introduced the treatment by peritendinous insufflation of filtered air, followed by massage. The operation, carried out with due regard to antiseptic precautions, may be performed in the standing or recumbent position. A tourniquet is applied to the forearm. The air drawn through iodoform gauze is slowly injected by means of a Potain's aspirator furnished with a fine needle, which is pushed into the subcutaneous tissues over the distension on the posterior line of the leg. When the air has penetrated the healthy, œdematous or indurated tissues of the strained part, the needle is withdrawn and the puncture closed with collodion. Next day the insufflated region should be gently kneaded to drive the air, which tends to spread eccentrically, into the meshes of the inflamed tissues. Massage is repeated morning and evening at the most distended parts and finally the leg is douched with cold water. By this method, Joly states that the effects of peritendinitis are rapidly and radically reduced, and that the indurated centre of tendinitis is quickly isolated and often reduced as well. In fifteen days, in certain cases, the tendon has become perfectly cool, clean, insensitive, and resistant to the effects of work. Insufflation is useless for old-standing extensive indurations of tendons, but for cases passing into the chronic state repeated injections followed by massage or counter-irritation may bring about functional recovery.

Chronic strain cannot be cured, though usually an attempt is made by further treatment to render the horse workable. With owners and veterinarians cauterization is the favourite remedy, but perhaps it is less popular than formerly, and it cannot be described as a specific for strained tendons. Superficial firing, in lines or by budding, when useful at all is most successful in simple cases, but of the many strained tendons fired in this manner the advantage to the horse has seldom been very marked. Deep line-firing probably is more beneficial than any other mode of cauterization, but whether it acts by inducing the formation of a more extensive subcutaneous œdema or by merely exciting cutaneous inflammation with consecutive compression of the strained part, is not known. Firing in points, the needle penetrating the induration, has a very limited application. Pyro-puncturing may be justified on the assumption that it reproduces deep-seated inflammation and establishes a further process of repair and consolidation of the distension. But unfortunately for the success of this method, it promotes the formation of adhesions between the skin and subjacent tissues, and shortening of the tendon, disadvantages

that detract much from its value as a remedy for strain. Besides, deep puncture firing is dangerous where a synovial sheath exists, and an operation that may produce open synovitis should not be undertaken without due consideration. For perforatus strain at the middle of the canon and strain of the suspensory, puncture-firing is useful enough.

Neurectomy.—When other treatment fails, neurectomy should be tried. Frequently it is too long delayed. It enables a horse that has become useless from chronic strain to work moderately for a year or longer according to the case. But, like other treatment, neurectomy has its limits of usefulness. It does not arrest the tendinitis, which after operation may become aggravated. It does not prevent, while it may hasten shortening of the tendon, and in some cases it does not entirely remove the lameness, which is said to be mechanical. It is not advisable for saddle horses, and for heavy draught horses, while permitting profitable disposal, it does not give satisfaction. Notwithstanding these objections, neurectomy should not be neglected in suitable cases of chronic strain.

CONTAGIOUS GRANULAR VAGINITIS IN COWS AND STERILITY.

Indexed B. A. I.

By STEWART STOCKMAN, M.R.C.V.S.[1]

Chief Veterinary Officer, Board of Agriculture, London.

I THINK it necessary to call attention to the above disease on account of its very contagious nature and the possible importance of its results.

The disease came particularly under notice during the course of the inquiry into epizootic abortion of cattle, and we have several times been consulted at the laboratory by members of the veterinary profession regarding it. It is not possible on the information available to express any very definite opinion about its prevalence, but our information is that it has become exceedingly frequent in certain parts of this country. It is not improbable that the disease has existed in Great Britain for many years, and that its apparently greater prevalence at the present time is due to a relation having been established between the more common but insidious chronic form without very evident external manifestations and the more noticeable acute form, which has led to the former being oftener

[1] From his Annual Laboratory Report for the past year.

looked for of late by manipulation of the genital passages, and owing also to the fact that it has lately been credited with causing sterility.

The disease is very prevalent on the Continent of Europe and in certain parts of the United States of America. In these countries it is said to produce epizootic abortion and be a cause of sterility in cows. With regard to vaginitis being a cause of epizootic abortion, we have excellent reasons, owing to the researches of the Board's Committee on the latter disease, for stating that abortion in this country is due to the bacillus of epizootic abortion. It is also established from the same source that sterility follows uncomplicated abortion in only a small minority of cases, and that the pure virus of epizootic abortion does not give rise to vaginitis when inserted into the genital passages. It seems not improbable that many Continental veterinarians have been led into error regarding the relation of contagious vaginitis to epizootic abortion, because both diseases are so exceedingly prevalent that they very frequently co-exist.

The inquiry carried on in Great Britain into epizootic abortion has certainly shown that it is not due to contagious vaginitis. With regard to vaginitis as a cause of sterility, it would appear from the writings of some distinguished foreign veterinarians that in their opinion the former is causally related to the latter. Some veterinarians are very positive on this point, and there seems to be no doubt that the two conditions may be found in association. That, however, is not necessarily sufficient to establish the relation of cause and effect between the two conditions, but I think one must grant, with fitting reserve made for the absence of accurate knowledge concerning the pathology of vaginitis, that there is some evidence to support the opinion that it may give rise to sterility. In connection with the inquiry into epizootic abortion in Great Britain, we met with herds which were reported by their owners to be affected with the latter disease, but which turned out on further inquiry to be affected with sterility, and no evidence of the existence of abortion was obtained. The sterility seemed to be gradually increasing amongst the females of the herds after the manner of a contagious but chronic disease. We possess practically no definite information regarding the prevalence and distribution of what for the moment might be referred to as enzootic sterility, and without wishing in any way to alarm the minds of stockbreeders unnecessarily, I think contagious vaginitis, and the very serious condition of sterility to which it is alleged to give rise, call for further investigation. It

has been possible to carry out a small amount of experimental and other inquiry in connection with vaginitis, but as the results merely touch the fringe, I do not propose at the present time to mention them in detail. I may say, however, that I have been unable up to the present to find any evidence that any of the various microbes isolated from the lesions is the cause of the disease, although it has been attributed to certain of them.

Symptoms.—The disease begins either in an acute or chronic form, but the acute runs into the chronic form, which usually lasts for months. In both forms the first symptom is redness and slight swelling of the external genital passage. In the acute form there is a certain amount of straining, and frequent passage of urine which seems to give pain. A mucous or muco-purulent discharge soils the external genital organs and the root of the tail. If the lips of the vulva be pushed apart the internal mucous membrane is observed to be very red in some parts, and covered at others by a greyish-yellow exudate. This form is probably what has for many years been known to farmers as "bull-burn." It is the insidious chronic form, however, to which I desire more particularly to draw attention. Although it may be ushered in by an acute stage, its advent may be unaccompanied by objective manifestations, such as a discharge. In fact, a cow may be suffering from chronic vaginitis and remain unsuspected by the most careful of attendants. The lesions, however, are very visible if one takes the trouble to expand the external genital orifice. The mucous membrane is redder than normal, and on various parts of its surface one sees a variable number of small granules about the size of a pin-head. At first they are red. Later they become paler, and look rather like small blebs, but they are not really vesicular; on the contrary, they feel hard and solid to the touch, and they may persist for several months.

Method of Infection.—The disease only affects cattle. If a swab of wool from the vagina of either an acute or chronic case be placed in the genital passage of a healthy cow, it is often sufficient to set up the disease in the latter either in the acute or chronic form, and the lesions may be evident in about five days after infection. From this it would appear that the bull probably acts as a mechanical carrier of infection. The bull is also said to become infected, and so act more surely as a disseminator of the disease. The bull, however, cannot be the only way by which infection is carried, for heifers which have never been to the bull are reported to suffer from the disease, and in this connection it is worthy of note that the condition

spoken of as sterility is also reported to be met with in heifers when they are first taken into the breeding herds. It seems probable enough that animals may contract the disease from contaminated bedding and drainage, but further investigation may provide more accurate knowledge concerning methods of infection. At the laboratory I have seen several animals become simultaneously infected owing to their temperatures having been taken in the vagina, and the source of infection was traced to an unsuspected chronic case which had had the temperature taken first.

Prevention.—The only preventive measures which can be suggested at present are isolation of the affected, continual disinfection of their genital passages, and of the litter, drainage, and flooring. The genital passage of each animal should be examined for lesions, and a special bull might be kept for those found affected. The hands should be carefully disinfected after manipulating the organs of an affected animal. It is advisable that the genital organs of prospective new purchases be examined.

With regard to the bull, disinfection of the external genital organs is indicated, and in districts where a co-operative bull is kept, the attendant should be instructed to examine all visiting cows for lesions of vaginitis before permitting service.

I hope it will be possible to conduct further inquiry into this disease, especially in relation to its alleged connection with sterility.

TRICHINOSIS.

By STEWART STOCKMAN, M.R.C.V.S.[1]

London.

Trichinosis is a disease of man and animals caused by a parasite called the *Trichina spiralis*. Human beings acquire the disease by eating the flesh of infested pigs. It is usually believed, however, that pigs in Great Britain are seldom carriers of the parasite. At least, if we are to judge from the number of clinical cases of trichinosis which occur in human beings in this country, it might safely be said that trichinosis of the pig is exceedingly uncommon. It is true, however, that several cases of slight infection of man have been found at *post-mortem* examinations in medical dissecting rooms, but owing to the small number of parasites found, the subject during life would probably never have felt anything more than slight

[1] From his Annual Report.

and local muscular pains, and the mildness of these attacks would depend on the small number of trichinæ in the flesh consumed.

So far as I am aware, there has been no outbreak of trichinosis sufficiently severe to attract attention in human beings in this country since 1871, when an outbreak occurred in Cumberland, and the pork, which was the cause of it, evidently contained a sufficient number of parasites to cause severe illness amongst the consumers.

The infected flesh contains larval *T. spiralis*, and when it is eaten by carnivorous animals the larvæ are set free from the muscle by the action of the digestive juices. When they reach the intestines they become adults and sexually mature. Each female gives birth to a large number of embryos, and the members of this second generation leave the bowel and are carried to the muscles in the blood stream. As one would expect, however, some of these larval forms are also excreted in the fæces of infested animals. The embryos, which invade the muscles, become coiled up and surrounded by a cyst wall formed by the tissues, and they may remain alive in this situation for a very considerable time. Animals may be infected not only by eating the flesh of other infested animals, but also by consuming other food which has been soiled by fæces containing the larval forms. It will be understood, then, how pigs may infect each other through their fæces. It seems probable, however, that the main factor in the up-keep of *T. spiralis* is the rat. These animals are very easily infected, and they are not infrequently in nature harbourers of the parasite. They may soil the food of pigs with their excretions while the parasites are in their intestines, or they may be eaten by pigs, and so give rise to disease in the latter animals.

In February, 1909, the Medical Officer of St. Thomas, Exeter, forwarded a portion of salted pig's flesh for examination to the Board, saying that on microscopical examination he had found in it what he thought to be the *T. spiralis*, and that a labourer, who had been consuming the flesh, had become very ill with what was thought to be trichinosis. On examining the specimen it was found to be badly infested with *T. spiralis*. One of the Board's Veterinary Inspectors visited the farm, and reported that the flesh had come from a sow which had been ailing for about two weeks before it was slaughtered. The sow was in good condition and fed well, but had shown great difficulty in using her hind quarters, so much so that she had even to be assisted to rise. On this account she was slaughtered, pickled, and used for food. Two people who consumed the flesh became seriously ill. The Inspector also reported that the farm premises were overrun

by rats, one of which he forwarded. The abdominal muscles of this rat contained a very large number of trichinæ. The rat was kept on ice for eight days, and was then used for experiment.

Experiment 1.—Small portions of the abdominal muscles were fed to white rat No. 1,018. This rat was found dead four days after eating the infested flesh. Adult trichinæ were present in the intestines, but no embryos could be found.

Experiment 2.—Intestines of rat No. 1,018 were fed to another rat No. 1,019. This rat also died four days after eating the intestines, and adult trichinæ were present in its large intestine. No embryos, however, were found.

Experiment 3.—White rats numbers 1,022, 1,023, 1,024 were fed upon the carcase and intestines of rat 1,019. The carcase was freely eaten, but the intestines appeared not to have been touched. Seventeen days after eating rat 1,019, numbers 1,022 and 1,023 were found dead. In the muscular part of the diaphragm of 1,022 encysted *T. spiralis* were found. Examination failed to reveal any parasites, however, in 1,023.

It is clear from the above observation that the muscles of the dead rat sent up from the infected piggery, contained living trichinæ. It also seems probable that the pigs became infested through the agency of diseased rats. It is, of course, a very difficult undertaking to keep a piggery free from rats. It is advisable, however, that pig-owners who cannot prevent rats invading their premises, should have the muscles of dead rats found on the place examined at intervals for the presence of trichinæ, since it seems to be the case that pigs in Great Britain, when they do become infected with this dangerous parasitic disease, owe the infection to the presence of infested rats. Amongst rats the disease is kept up not only by embryos from the intestines contaminating their food, but also by the cannibalistic habits of these vermin.

SPIROCHÆTÆ IN LESIONS AFFECTING THE PIG.

By J. A. GILRUTH, M.R.C.V.S., F.R.S.E., D.V.Sc.

Professor of Pathology and Bacteriology, Veterinary School, Melbourne University.

Indexed B. A. I.

Skin and Subcutaneous Lesions.

The presence of these protozoan parasites have been already described as found in certain pathological conditions of the pig, notably by Dodd, as occurring in an ulcerative skin disease in the Transvaal,

transmissible by both contagion and by inoculation (*Journ. Comp. Path.*, 1906), and by Cleland in "castration tumours" of the pig in West Australia ("Parasitology," vol. i., No. 3).

Recently the occurrence of spirochætes have been observed by me in several lesions of somewhat diverse character in Victorian pigs, of which the following is a short description:—

In January, 1910, a pig's head was received from the Veterinary Department of the Victorian Government. It was affected with a large ulcerating tumour, the size of a fist, on the side of the cheek. The tumour was dense and had the characteristics of a fibroma. Unfortunately, no preservative having been used, putrefaction was somewhat advanced.

Microscopical examination showed throughout the structure of the tumour numerous spirochætes, the majority similar in size to those found in the fowl, but a number were much shorter than those usually found in avian blood.

As the owner advised that he had previously had three pigs similarly affected, and still had one showing a similar condition, Dr. Cameron requested him to forward it alive. It was duly sent, but unfortunately arrived during my absence on a holiday.

On arrival it was observed to be suffering from a hard swelling on the side of the left jaw, and a healthy young pig was placed in the same pen to test the possibility of contagion.

On my return to Melbourne it was found that the swelling on the left jaw had disappeared, but that the left knee was swollen and showing a small ulcerating surface just under the joint. Below this swelling was a caseous and necrotic area extending downwards about 2 in. and surrounded with chronic inflammatory tissue.

Smears from this showed numerous spirochætes and a mixed bacterial flora.

Attempts to transfer the condition by inoculation on scarified skin resulted in the production of a number of granulomatous, inflammatory chancroid lesions being slowly developed at, and in the vicinity of, the scarified area. These ultimately disappeared, but at no time were spirochætes detected, although microscopical examinations were frequently made. The granulomatous tissue appeared to be due simply to streptococci, which were present in large numbers.

The contact pig remained normal.

Recently I have had an opportunity of examining two cases of scrotal tumours similar to those described by Dr. Cleland, through the courtesy of Dr. John Robertson, Director of the City Abattoir.

Both pigs were in fat condition, about 18 months old, and I was informed the viscera of each were normal. At first glance the tumours had almost the appearance of normal testicles somewhat enlarged, being very prominently situated in the scrotal region subcutaneously. The skin was normal but for the scar where the wound by the castration knife had been made. In one case a tumour was situated in each scrotal sac, but in the other only one was present. On dissection the tumours, which were ovoid, slightly flattened, and of the diameter of a large orange, were found to be circumscribed, and fairly dense in consistency. On section the new growths were seen to be composed of fibrous but œdematous new connective tissue, enclosing a central irregular necrotic caseous area almost the size of a walnut, immediately around which the fibrous tissue was distinctly of a greyish dirty colour, strongly contrasting with the translucent homogeneous appearance of the peripheral mass.

In each case the spermatic cords at the distal end of which the tumours proper were situated were thickened, being about 1 in. in diameter, and contained several circumscribed caseous areas, varying from the size of a marble to that of a walnut.

Microscopical examination of smears of the central degenerated material from the terminal tumours showed many spirochætes similar to those already described, along with masses of various kinds of micro-organisms, such as cocci, long and short bacilli. Scrapings from the œdematous fibrous tissue showed also many spirochætes with numbers of bacteria, though few in comparison with those present in the caseous centre. The caseous nodules in the thickened cord while containing many mixed bacteria appeared to be free of spirochætes.

As to whether the spirochætes were the cause of the new fibrous growths under consideration is a question that requires further investigation, but the indications at least are that their presence conduces to the formation of the new fibrous tissue observed, while the central degeneration is probably the result of the bacterial invasion.

Sub-mucous Cysts of Large Intestine.

Spirochætes have been found by me recently associated with intestinal lesions of the pig, but apparently they were not the cause of any serious general disturbance.

The lesions were first observed in two young pigs received alive from the country for examination. The animals on arrival were very

lean, and though the temperatures were above normal, the appetite was good. The blood of each was normal, so far as erythrocytes were concerned (7,500,000 to 8,000,000) but there was a definite increase in leucocytes (50,000), chiefly eosinophiles.

During the succeeding three days, as the condition was rapidly improving and no definite symptoms could be detected beyond a slight fluctuation in the temperature, one was slaughtered for examination, and the other a week later, when it was obvious a very decided improvement in appearance had taken place, these facts alone pointing to some neglect or dietetic error having been the cause of the poor condition.

The first pig killed showed a definite pathological condition of the large intestine. The mucosa of the cæcum was affected for about an area of eight inches with patches of inflammation covered by diphtheritic false membrane. The large intestine throughout its whole course showed numerous circular, flattened, greyish nodules, each about the size of a small pea, there being about two to four present to the square inch. These nodules were distinctly observable without incising the bowel, and caused some projection of the serous covering. On examination of the mucous surface slight circular elevations were observed corresponding to these nodules, the majority showing a minute central depression, through which the contents could be readily squeezed. These nodules were apparently cystic in nature; in some instances the contents appeared translucent, jelly-like, and not readily broken down, with a small greyish, caseous centre; in others the contents were completely caseous.

On microscopical examination the material was seen to consist chiefly of fibrinous *débris* with pus cells and some columnar epithelia. A peculiar feature of the less degenerated contents was the presence in smears of a finely laminated membranous structure, as if part of a parasitic cyst wall. Stained by Giemsa's method myriads of beaded bacteria of varying length, cocci, and bacilli could be detected, but, in addition, especially in the "laminated" membrane, could be seen many delicate spiral organisms with all the characters of spirochætæ. These were so regular and so numerous in the "laminated" structure which was comparatively free of bacteria that there seemed to be some decided connection. It should be observed that in the fresh state no definite movements of the spirochætes could be observed, and that there were always some cercomonas, actively motile, found present.

Sections of the intestinal wall demonstrate these nodules to be of

the nature of small cysts of the deeper glands, external to the muscularis mucosæ, and apparently all are more or less affected. Considerable irritation is evidenced by the accumulation of lymphocytes and some formation of new fibrous tissue, with atrophy of the muscular wall, at the periphery, while the centre is composed of fibrinous material with shed epithelial cells and more or less degenerated polymorphs. Masses of bacteria, chiefly bacilli showing great irregularity in staining, can be seen present amongst the cells and *débris*.

It may be noted that a young pig about six weeks old, which was placed in the same pen as these pigs, developed a high temperature with capricious appetite ten days later, and was killed on the fourteenth day, as it was evidently recovering from this condition. *Post-mortem* examination disclosed a broncho-pneumonia with irregular areas of degeneration (caseous) which was evidently the cause of the indisposition. In addition, however, on examination of the mucous membrane of the large intestine a number of nodules, similar but smaller in size to those above described, were observed. They were not visible on the serous surface and none were degenerated; all showed the central depression and contained a gelatinous material, amongst which bacteria as before, and a few cercomonas, could be demonstrated, but no spirochætæ.

That these nodules were not induced by contagion from the previous cases was proved by the fact that a control from the same litter which had remained in good health, killed ten days later, was also affected with similar nodules. These again contained no spirochætes, but many bacilli and a few cercomonas.

It may be assumed, therefore, that whatever may be the pathological significance of these nodules or cysts (obviously not great), which were probably due to the bacterial invasion, the presence of spirochætes within their contents was probably accidental.

In neither of the first two pigs were any metazoan parasites observed, though careful search was made throughout the whole of the intestinal tract. It may therefore be concluded that the definite eosinophilia present in each was probably due to the spirochæte invasion of the intestinal cysts described.

NOTE ON THE EXISTENCE OF SPIROCHÆTOSIS AFFECTING FOWLS IN VICTORIA, AUSTRALIA.

Indexed B. A. L.

By J. A. GILRUTH, M.R.C.V.S., F.R.S.E., D.V.Sc.(Melb.).

The fowl tick (argas) has been recognized as an exceedingly troublesome skin parasite of the domesticated fowl in certain parts of the northern districts of Victoria for a number of years past, and its association with a febrile condition, especially in young birds during the summer months, has, it would appear, been commonly observed, although as far as I can gather, beyond some short paragraphs in the daily press (until the last month when an article by Dr. A. A. Brown appeared in the *Journal of Agriculture*), no definite scientific observations have been placed on record. This is rather surprising in view of the fact that the Deparment of Agriculture, I am informed, has for some years been endeavouring to prevent the spread of the tick to other districts.

Soon after my arrival in Melbourne at the commencement of last year I was informed of the existence of the "so-called tick-fever" in certain districts. Seeing that since 1903, when Marchoux and Salimbini first described the presence of a spirochæte in the blood of Brazilian fowls affected with the tick argas, other observers in India, Sudan, Rhodesia, Bulgaria, and elsewhere have also demonstrated the connection between the two parasites, I was naturally anxious to ascertain if in Australia, along with the tick as a skin parasite, the spirochæte was also associated as a blood parasite. I found it, however, impossible to secure a tick-infected fowl during the past summer.

Since then Dr. S. Dodd, Chief Veterinary Officer and Bacteriologist to the Queensland Government, in his last annual report describes fully the disease Spirochætosis as affecting Fowls in that State, and has demonstrated its transmission by the common fowl-tick *Argas persicus*.

Through Dr. Brown of the Agricultural Department at the request of the Minister for Agriculture I received on January 22 a live fowl presenting the following definite symptoms: General dejection, somnolence, ruffled feathers, comb pale, slight diarrhœa, and loss of appetite. Only four ecto-parasites were to be found on the skin and these were apparently all larval forms of a parasite of the argas type, and were handed to Dr. G. Sweet for identification. Dr. Swift has described these as belonging to a new species *A. victoriensis*.

Examination of blood smears made in the usual way, fixed in alcohol and stained with Giemsa's stain, gentian violet, &c., demonstrated considerable numbers of the characteristic spirochætæ as described by Marchoux, Laveran, and others. There was also a marked increase in the number of eosinophile white blood corpuscles.

The spirochætæ increased in number till the time of death fifty-six hours after arrival. *Post-mortem* examination did not disclose any decided pathological change, and the spleen was not enlarged.

Inquiry from Dr. Brown elicited the information that the bird had been sent from a non-infected to an infected district, and there exposed to the ticks only six days prior to being forwarded to me, which indicates the rapidity of the infection.

A live fowl was inoculated subcutaneously with 5 drops of blood from the heart of the first fowl a few minutes after death. No swelling developed at the seat of inoculation. Spirochætes were found in the peripheral blood on the third day but only in one to every twenty fields of the microscope. On the fourth and fifth days the numbers increased greatly and several could be seen in each field. On the sixth day, however, extremely few could be detected, while subsequently none could be seen. The animal remained in normal health. The disappearance occurred without any preliminary clumping and no intracorpuscular bodies of Balfour's "after phase" could ever be determined.

Careful examination of smears from the naturally infected fowl, it should be noted, failed to detect any bodies within the red blood corpuscles such as described by Dr. A. A. Brown in a recent number of the *Victoria Journal of Agriculture*.

Intracorpuscular forms of spirochætes have been described by Balfour [Third Report, Wellcome Research Laboratory, Sudan] as constituting the "after-phase" of spirochætosis. These bodies were only found in animals which recovered, and he regards them as a definite stage in the life history of the blood parasite. Von Prowozek has recorded somewhat similar intracorpuscular bodies in fowl spirochætosis. Dodd, however, in his Queensland experiments failed to demonstrate such bodies in any of the recovered fowls. My examination of the blood, in which the spirochætes disappeared so suddenly also failed to detect any similar bodies to those described by Balfour.

Clinical Articles.

HYGROMA—BIER TREATMENT.

By J. J. O'CONNOR, M.R.C.V.S.

Professor in the Royal Veterinary College of Ireland, Dublin.

Subject.—A valuable brown, weight-carrying hunter gelding, aged 5.

History.—A little over twelve months ago a small postero-internal swelling appeared just below the knee, without causing lameness. It was blistered without effect, and the swelling had been increasing in size ever since. Three or four veterinary surgeons had seen it and considered it best to leave it alone.

Symptoms.—A swelling of the same consistency as a chronic bursal enlargement with fairly thickened walls in which fluctuation could be detected, situated just below the knee at the postero-internal aspect of the limb, involving the inner aspect of the flexor tendons; painless to the touch and not causing lameness. Its size was about that of a hen's egg. Pressure on the swelling did not cause a distension to the outer aspect of the tendons but caused a bulging above and behind the knee, not in the position of distension of the carpal sheath.

Diagnosis.—A hygroma or cystic collection of serum beneath the skin, or possibly a distension of portion of the carpal sheath which became partitioned off from the remainder.

Prognosis.—The swelling might continue to increase in size and eventually interfere with free flexion of the knee. Blistering or line firing not likely to be beneficial, but needle point firing might have a good effect. Opening, evacuating and injecting with tinct. iodine likely to have good result.

Treatment.—Having explained to the owner the possible danger of opening the carpal sheath, and had the animal insured, cast him, injected cocaine locally, and opened the swelling at its lowest point. A quantity of blood-tinged serum escaped. On exploring the cavity it was found to extend for a distance of above 3 in. above the lower end of the radius, and its lining was found to be infiltrated with calcareous material. I injected the cavity with tinct. iodine and inserted into it a pledget of gauze saturated with tinct. iodine. The subsequent treatment consisted in syringing with tinct. iodine, but after three weeks of this treatmeut the cavity showed no signs of healing, and its orifice assumed an ulcerous appearance. I then rubbed powdered zinc sulphate all over its lining. This caused pain, lameness

and swelling which disappeared after a few days, but still the sinus persisted. I tried iodine again for some days without avail, and afterwards injected 5 per cent. zinci. chlor., which also failed to have the desired effect. I was afraid to use hydrarg. perchlor. in powder for fear of causing a too deep slough, and thus exposing the carpal sheath. I felt sure that opening up the passage would be followed by healing, but I did not wish to make a large wound at the back of the knee; consequently I resolved to try the hyperæmic treatment. I applied a rubber tourniquet above the knee fairly tightly but not so tight as to arrest the arterial circulation, and left it on for three hours, repeating this for two days in succession. This treatment caused a large inflammatory swelling about the knee which was very painful, making the animal extremely lame. I then allowed a couple of days to intervene before applying it again, and then left it off until the swelling subsided, when I found that the passage had become smaller, seeming to have closed in at the sides, and that the discharge was considerably diminished. I then allowed the horse to be taken home and turned out to grass, instructing the owner to apply the band occasionally in the way I demonstrated to him. The owner informed me afterwards that he used the band, he did not say how often, perhaps once or twice, and that the condition continued to improve after he had taken the horse home, and the cavity soon closed up altogether. Considerable thickening remained, but the horse went sound again, and the cystic condition was got rid of. The case was disappointing as it was three months under treatment, but it showed the efficacy of the Bier treatment after other methods had failed.

CYST IN THE SINUSES OF A COLT.

By Professor J. J. O'CONNOR.

Dublin.

Subject.—A strong yearling cart colt in good condition.

History.—Six months previously to my seeing the colt he had a slight discharge from the nose which the owner thought was the result of a cold, but it persisted, and the following symptoms gradually developed.

Symptoms. — A fairly copious mucoid discharge from the right nostril, a loud respiratory noise, a large swelling over the frontal and superior maxillary sinuses, and over the level of the fangs of the superior molars, and a thin-walled cyst in the right nasal passage,

which could be felt with the tip of the finger passed in through the nostrils. Dulness was not evinced on percussion over the sinuses.

Treatment.—Cast the colt. On examining the cyst in the nose it burst and discharged a reddish glairy fluid. I made three trephine openings, one into the uppermost part of the frontal sinus, one into its lowest part, and one into the superior maxillary sinus. The same kind of fluid as was present in the nasal cyst escaped from the sinuses, it was inodorous. I washed out the sinuses with an antiseptic lotion. The subsequent treatment consisted in irrigating the sinuses daily with an antiseptic lotion, chinosol or lysol, the openings being prevented from closing by pledgets of gauze. After about a fortnight of this treatment the condition seemed little improved except that the breathing was not so noisy, on account of the cyst in the nose having ruptured. I therefore cast the animal again, and made all the openings much larger by breaking away pieces of bone with the bone forceps and chisel and mallet, so that I had a clear view of the interior of the sinuses, a great part of which was occupied by loculi composed of soft spongy bone and embryonic fibrous tissue, which I removed with the curette. I then packed the sinus with gauze saturated with tincture of iodine. Afterwards daily treatment consisted of flushing out the sinuses with lysol solution, irrigating with tincture of iodine, insufflating with iodoform and boric acid, and plugging with gauze wrung out of tincture of iodine, pure or diluted with two parts of water. Under this treatment the condition gradually improved, and after four or five weeks the lining of the sinuses assumed a pink, healthy appearance, the discharge ceased, the swelling on the face greatly subsided, and the respiratory noise disappeared. The case appeared to be cured, the holes were allowed to close, and the colt was sent home to the country. Three months afterwards the owner said he was doing well.

Eight months after his discharge the colt was sent to me again, showing the same symptoms as before, and I opened the sinuses in the same places and found them quite full of the same kind of fluid. In addition, I made an opening into ths nose from the lower part of the frontal sinus. On the second day after the operation the animal showed all the symptoms of acute continuous colic, and in spite of the usual treatment he died in the course of a few hours. *Post-mortem* examination was disappointing as regards revealing a cause for death. The only abdominal abnormality found was a small chronic abscess in the mucous membrane of the stomach, and

slight impaction of the colon. There was no inflammation of the brain or meninges. The whole carcase became rapidly emphysematous after death.

A DISEMBOWELLED CALF; OPERATION AND RECOVERY.

By E. RYAN, M.R.C.V.S.

Strokestown.

I was called one morning to a case of parturition in a Shorthorn cow, and upon arrival found the owner had just removed the calf, breaking off the umbilical cord so close to the abdominal wall that the latter had been torn rather badly. The wound thus made was fully 5 in. in length, and a bunch of intestine, quite as large as a football, was protruding. As the calf was lying on the floor of the byre, and this was very dirty, they were already much soiled, so that I thought that by far the better plan would be to destroy the beast at once. At the request of the owner, however, I decided to try and return them. After washing with lysol and water I got them back into the abdomen and sutured the wound with a continuous suture of chinosol tape. The bowels were very much soiled, and I am quite sure that I did not get all the material off, but to my agreeable surprise the calf made an uninterrupted recovery. In fact, it behaved in such a way that had I not known of the accident I should never have suspected, when I saw the calf a couple of days later, that anything had ever happened to it.

A CASTRATOR'S ERROR.

By JNO. L. PERRY, M.R.C.V.S.

Cardiff.

In the early part of September, 1908, I received a letter asking me to attend a cart horse, aged 3, upon which an attempt at castration had been made by an unqualified man three days previously.

The owner said in his letter: "When the operation was done, I was present and saw one testicle taken away with clam and hot iron as usual. The castrator, a man who does all that kind of work about here and has hitherto been most successful, then 'bunched' up something in the clam. I saw, at once, it was not a testicle, and told him

so. He, however, persuaded me that the colt was malformed, and that it was the other testicle all right. I, however, left in disgust, and learnt afterwards that he had at once proceeded to sear through this 'something' with the hot iron. Immediately this was completed about 12 in. of penis fell from the horse's sheath to the ground. So he had amputated the penis in mistake for a testicle! Being thus convinced of his error he then found and removed the other testicle. The horse is now very weak and eats but little, his sheath is a tremendous size, like a sack of potatoes."

I was away from home at the time, so Mr. C. E. Smith, M.R.C.V.S., saw the horse in my stead. He found the sheath almost justified the description given it by the owner of the horse. It was engorged and pointing in places with infiltrated urine. After casting the animal and well lubricating the inside of the sheath with vaseline, he discovered, after a lot of tedious manipulation, the mutilated end of the penis about a foot away from the natural opening of the sheath. The swelling being so severe, the urine could only come away in a small dribble, so he decided to make an opening for the penis stump to come through the sheath in a position close to the proper castration wounds. The urethra protruded about $\frac{1}{8}$ in., but it was impossible to get a skin attachment for it: so it was left as it was with the intention of completing this part of the operation later on when the swelling had subsided. Punctures were made in various parts of the sheath to allow the urine which had infiltrated into the surrounding tissues to drain away. All the parts were thoroughly cleansed with warm antiseptics and then dressed with carbolized vaseline.

All this took a long time, and when the horse was allowed to rise he was terribly "done up," but soon revived after a stimulant in the shape of half a pint of whisky, which, by the way, the owner parted with very reluctantly. As the farm was twenty-seven miles away, the owner said he would send word as to the horse's progress. This he did, saying there was "daily improvement."

I saw the case myself ten days afterwards. The sheath was slightly swollen; horse eating and improving in condition. Standing behind him and pulling the tail aside I could see about 4 in. of penis hanging through a wound in the sheath, and in position just where a mare's teats would be. The penis pointed downwards and backwards, and when urination took place there was a stream about the callibre of a clinical thermometer case directed upon the points of the hocks. The urethral opening was clearly diminished in lumen, and I told the owner that the horse should be cast again, and a further small portion

of the penis removed so that the urethra could be properly everted and stitched back to avoid a further stricture. This he would not consent to, preferring to "wait and see" how the horse went on.

I was not asked to attend the horse again, but being in the locality a month or two afterwards saw him at grass. Both hocks were then in a terrible mess, due to the constant dribbling of urine upon them. The urethral opening was evidently very small, as one could see the urine coming away from the penis in a very fine spray. Owner still refused surgical interference. I wrote him about twelve months ago on another matter, and asked him how "Farmer" was going on, expecting to hear he had been sent to the kennels. His reply was, "Horse working on farm regularly, and except for requiring an occasional drench goes on all right."

I might add that I tried at the time to persuade the owner to institute proceedings against the castrator, either for cruelty or in a civil court, but this he would not do, the reason being, as I learnt afterwards, that he had arranged terms for the castrator to pay him the sum of £25 in instalments, as damages. This would, of course, account for his desire to avoid further expense or publicity. He wanted the matter kept quiet till the money was paid; hence also his employing me in lieu of other veterinary surgeons nearer his home.

ADENO-CARCINOMA OF THE LUNG, WITH SECONDARY GROWTHS, IN A COW.

By J. A. GILRUTH, D.V.Sc., M.R.C.V.S., F.R.S.E.

Professor of Veterinary Pathology, Melbourne University.

An interesting report of this case, along with specimens for microscopical examination, was received from Mr. W. T. Sabin, Government Veterinarian, who is most assiduous and careful in noting unusual pathological conditions. The animal was a Jersey cow, aged 8.

History.—The cow, then in good condition, was attacked with milk-fever after calving, in November, 1907. She recovered under treatment, but remained dull and weak. Atrophy of the muscles of the forelimbs developed, but this was considered due to injury received when she was suffering from milk-fever, it having been deemed necessary to drag her some distance to a convenient and sheltered place. Her milk, which was abundant, was, however, given to her calf, as it was obvious the cow was not in health. Later on,

tuberculosis being suspected, she was tested by Mr. Kerrigan, but there was no reaction; the result, nevertheless, was considered doubtful, as her initial temperature had been abnormally high. Finally, general weakness became so marked that she was unable to rise, and the owner destroyed her as hopeless on July 22, 1908.

Post-mortem Appearances.—A *post-mortem* examination was made by Mr. Watt, Assistant Meat Inspector, who, finding unusual lesions in lungs and kidneys, communicated with Mr. Sabin, and he, after visiting the place and carefully examining the affected organs, reported as follows:—

The general condition of the animal was poor. The lungs showed no growth externally, but the left lobe was enlarged by a visible swelling. On section of the lung a dilatation of a bronchus was opened, and a portion of new growth became visible, forming part of the wall, appearing as a smooth yellowish surface, not ulcerated, but apparently covered by normal mucous membrane. The swelling was found to be due to a tumour with circumscribed periphery, ovoid in shape, about 5 in. long and 3 in. in its narrow diameter, fairly dense, and fleshy in consistence. The appearance of the cut surface was yellowish, with irregular whitish fibrous bands throughout, and here and there areas of degeneration (colloid). Scattered through the substance of both lungs were smaller, more spherical, areas, probably secondary, which were homogeneous and yellowish on section. The mediastinal gland was enlarged, about two-thirds of its substance being occupied by a new growth similar to the secondary tumours in the lung. The kidneys were also metastatically affected. In one were several globular tumours from 2 to 3 in. in diameter, coalescing at points, and numerous smaller areas, varying from the size of a pin-head to that of a pea, the greater part of the normal tissue of the organ being replaced by the new growths. The other kidney was also affected, but the tumours were fewer in number, about half the organ being replaced, however, by the new growth. The larger tumours were visible on the surface as slight nodular projections of a yellow colour. It was not until section had been made that the full size of any nodule was disclosed. The consistence was fairly dense, the colour a homogeneous pale yellow, and almost caseous-looking. Each nodule was spherical, and definitely circumscribed by a thin, fibrous false capsule.

Mr. Sabin formed the opinion that the disease was a form of cancer, the large tumour in the lung being the primary growth, the others, as well as those in the mediastinal gland and the kidneys, being secondary. The fact that the large tumour of the lung alone

showed on section visible bands of fibrous tissue, and also areas of degeneration, supported the conclusion; and microscopical examination of sections confirmed it as far as was possible.

Microscopical Examination.—The large new growth in the lung presents under the low power a distinctly lobular appearance, similar to that of a secreting gland. The lobular areas are irregular in size and contour, and are separated by bands of fibrous tissue. Under higher powers these lobules are observed to be comprised of more or less cubical epithelial cells, generally arranged as solid processes, of irregular thickness, aud sometimes branching, but here and there they line as a single layer of cubical cells very definite acinous or alveolar spaces. The fibrous stroma is comparatively scanty throughout the new growth, unless in the bands dividing the larger lobules, or groups of lobules, and at the periphery of the neoplasm where it forms a a false capsule.

The secondary growths found in the lung and mediastinal gland do not show so definite a lobulation, though it is still present. The general arrangement of the epithelial cells, including the occasional alveolar distribution, is, however, similar.

The secondary growths in the kidney show no lobulation, and no alveolar formation. There is little stroma. At the periphery there is a distinct false capsule, formed by a new fibrous tissue, which is seen invading the parenchyma, causing atrophy and marked distortion of the glomeruli and the tubules, but there is no infiltration of the renal tissue proper by the new epithelial growth.

AN INTERESTING SPLEEN, THE RESULT OF STRANGLES.

By JOHN VARNEY, M.R.C.V.S.

Winslow.

Subject.—Bay gelding (hunter), aged 4, 16.1 in. height. Examined for soundness May 4, and passed sound, in the course of a few days he commenced to cough, with nasal catarrh and acute laryngitis, accompanied with a very high temperature (106° F.). With the usual treatment the animal apparently recovered in three weeks, showing no symptoms of complications or sequelæ. The horse was ridden and did his work for a fortnight.

May 25.—I examined a chestnut gelding (arrived on the same premises a day or two previously) for soundness, but I found he had

a slight discharge from the nose, accompanied by a sore throat, and knowing that the other horse had been so ill, I held the certificate over. This horse developed similar symptoms to the other horse, and recovery took place in about three weeks. At the same time a yearling developed similar symptoms, accompanied with strangles; this animal recovered.

June 15.—Another horse, a bay hunter gelding, was observed to have a cough, with nasal discharge, acute laryngitis, temperature 104° F., and slight swelling of the submaxillary glands. In the course of three weeks five or six abscesses formed and broke, and discharged in the usual manner, in the submaxillary and parotid region. This case, I consider, was remarkable for the enormous quantity of pus, mucus, and saliva which continually came away for at least three weeks, the manger, rack, and boards having to be cleared off and disinfected three times a day; in fact, it was so great that we removed the horse to an isolation box.

After July 5 the discharge appeared to decrease, and the horse appeared to be on the way to recovery, as no other abscesses showed themselves. However, he did not improve to my satisfaction in the next week, the temperature rising and falling, more discharge some days than others, and the whole time losing flesh and condition, although milk and eggs were taken freely three times a day, also small quantities of old hay, new hay, and vetches, and he would eat about a quart of crushed oats and bran with chaff in the twenty-four hours. However, debility and emaciation increased, so that the horse could hardly walk about, temperature during this time being a little over 102° F., and the pulse 60.

July 22.—Horse much worse. Temperature 104° F., pulse 75, very weak, slightly accelerated breathing, and other symptoms pointing to a fatal termination, and, as I had informed the owner some fortnight or three weeks ago, the probability of an abdominal abscess.

I should like to say that, during the whole of the horse's complicated illness, pulmonary symptoms were never predominant or asserted themselves above others, the lower portion of both lungs appeared consolidated, and applications of strong liniment had been applied to the throat two or three times. I had remained in the box with this horse several times alone from ten to fifteen minutes, and particularly noticed him lifting up first one hind leg and then the other towards his belly, with a corresponding twitching movement of his tail, indicating slight abdominal pain, afterwards throwing his head right up with a groan and deep sigh; moving listlessly round, and going

through the same symptoms again, after which he would commence to feed again. He often lay down; there was no trouble with the bowels or kidneys except the last few days, when the urine appeared almost black, but in fair quantity.

The appearance of the patient became markedly altered; the face was drawn and haggard, the whole expression being anxious, and the skin distinctly "hidebound." At this stage he was seen in consultation by Professor Hobday, who, thinking that there was fluid in the chest and consolidation of lung tissue, advised an exploratory puncture with trocar and cannula. This was done on each side in the usual situation, but only a small quantity of blood-stained fluid followed, and within three or four minutes the horse collapsed and died quietly, almost without a struggle.

The *post-mortem* examination was most interesting, especially in regard to the organs in the abdominal cavity. The spleen was enormously thickened and enlarged, weighing 37 lb., and being adherent to the abdominal wall, to the diaphragm, and to the stomach. Between its upper end and the stomach there was a strangles abscess almost as large as an ordinary football, containing very offensive pus, and in the front portion of the thorax there was another abscess as large as a fair-sized pine-apple. The lungs were congested and consolidated in places, and there was a small quantity of blood-stained fluid in the chest.

Remarks.—The case is of particular interest on account of the enormous size of the spleen, the size of the other internal abscesses, and the way the poor beast lingered on under such conditions without exhibiting worse constitutional symptoms.

OVARIOTOMY IN A MARE.

By J. J. O'CONNOR.

Professor in the Royal Veterinary College of Ireland, Dublin.

Subject.—A good bay cart mare, aged 9, in good condition.

History.—The mare was an inveterate kicker in harness and eventually the vice became so bad that she could no longer be used. As the result of the constant kicking her tail became badly damaged against the front of the cart, being affected with a large suppurating contused wound.

Treatment.—Having thoroughly disinfected the tail and vagina, removed the diseased portion of the tail with the docking knife, I

performed ovariotomy. The result was very satisfactory, as the mare afterwards became perfectly quiet, working steadily in harness without kicking.

FIXATION OF THE PATELLA IN A HEIFER.

By Professor J. J. O'CONNOR.

Dublin.

Subject.—A six-months-old Jersey heifer.

History.—Had been found one day lame in the near hind leg, which was carried extended backwards.

Symptoms.—Extension of the stifle and hock joints of the near hind limb, the toe barely touching the ground. The patella was firmly fixed above the trochlea of the femur.

Treatment.—(1) Tried in vain to dislodge the patella by external manipulations. (2) Cut the internal lateral ligament of the patella, an operation which Cadiot says always permits of reduction, but in this case it was without effect. (3) Cut the vasti and rectus femoris muscles at their insertion into the patella without having any effect on the latter, which seemed anchylosed in its new situation. But this operation improved the gait considerably, the animal being able to put her foot flat on the ground.

AN INTERESTING MONORCHID.

By FREDERICK HOBDAY, F.R.C.V.S.

Kensington, W.

The patient, a chestnut cart gelding, 2 years old, was one of three cryptorchids presented for operation, in consultation with Mr. T. Stainton, M.R.C.V.S., of Reading, on June 14 last. All were abdominal "rigs," and all showed peculiar features. The testicles in the other two cases were found and removed, each eventually making an uneventful recovery, but the chestnut was of especial interest as it proved to be a true monorchid. There was no evidence or history of any prior attempt at castration; in fact, it was known with certainty that no testicle had ever been removed. The left one was present in the scrotum and was removed without any trouble. On the right side the abdomen was penetrated in the usual situation, close to the inguinal ring, and a careful search revealed not only the absence of testicle, but a gradual merging of the end of a rudimentary cord into the lining of the peritoneum of the pelvis. After making sure

of this fact, by tracing it repeatedly, the hand was withdrawn and the inguinal canal carefully closed by sutures. The colt was allowed to come out of his chloroform, and got up apparently none the worse for his experience. This was about 5 o'clock. At 10.15 p.m. the animal was heard to be making a noise in the box as if in violent pain, and upon examination the bowels were found to have descended. The weight had ruptured one of the sutures and a loop of bowel had come down nearly as far as the hocks. Assistance was summoned, and after considerable difficulty the colt was cast and the bowel returned. As much washing and disinfecting was done as was possible under the circumstances, and a plug of cotton wool was inserted. This was inserted (according to Mr. Stainton's idea) in a very ingenious manner underneath a row of sutures, and then followed by a second row of sutures, in such a way that the pad could be changed without danger of allowing the bowel to escape, the first layer of sutures not being touched or interfered with in any way.

On the following morning the colt's temperature was 103° F., and during the subsequent days it varied been 102° and 103° F. The pad of cotton wool was changed on numerous occasions, and febrifuges, tonics, or stimulants were administered internally at discretion. Antistreptococcic serum was also given.

Peritonitis was evidently present and in spite of all efforts, death eventually took place on July 15.

A *post-mortem* examination at which Dr. Kendall, D.V.Sc., M.R.C.V.S., and Mr. Benson, M.R.C.V.S., in addition to Mr. Stainton and myself were also present, confirmed the absence of any testicle on the right side, nor was there any evidence of such an organ ever having existed, the spermatic cord being clearly traceable and merging imperceptibly into the peritoneum of the pelvis. Such cases are rare, and are worth recording. I have already reported a similar case in my little brochure upon "Cryptorchid Contraction," and a further still more curious point in which both testicles were absent.

The remainder of the *post-mortem* examination was only of interest in connection with the peritonitis. The loop of bowel which had descended was matted together, and there was a long abscess between the two portions of the loop. This contained a piece of dirty straw which must have been overlooked, as it quite readily might have been, when the bowels were washed and returned.

On the left side the end of the cord from which the testicle had been removed could be found quite easily, and had nothing about it upon which to make any comment.

Canine Clinical Notes.

DENTAL FISTULA IN A DOG.

By Professor J. J. O'CONNOR.

Dublin.

Subject.—A Skye terrier.

Symptoms.—A dental fistula communicating with the upper carnarsial tooth on the left side.

Treatment.—In extracting the tooth, which was firmly fixed in its socket, the anterior fang fractured, and portion of it remained in the alveolus. As this fang seemed healthy it was thought that the fistula might now heal, but it refused to do so, and consequently I cut through the mucous membrane at the junction of the gum and cheek, and separated the skin and underlying tissues from the bone at the level of the alveolus, which I then trephined, and removed from it the remaining portion of the dental fang. After the treatment the fistula rapidly healed.

VENTRAL HERNIA IN A BITCH.

By Professor J. J. O'CONNOR.

Dublin.

Subject.—A valuable harrier bitch.

History.—The bitch was found in the kennel with an abdominal swelling, apparently caused by being attacked by the other dogs.

Symptoms.—Those of a large hernia in the middle line a little in front of the pubis.

Treatment.—I opened the hernial sac and found that it was not lined by peritoneum, and the omentum was attached to it throughout. The edges of the hernial ring, which admitted two fingers, were ½ in. in depth or thickness, and very hard. Hæmorrhage was more profuse than usual in this region. I separated the adherent omentum from the sac, removed some of it, and sutured the hernial ring with silk. The insertion of the sutures was very difficult with a slightly-curved needle, owing to the thickness and hardness of the lips of the hernial ring. I shortened the skin of the sac and sutured the skin wound. A good deal of blood oozed through the cutaneous sutures, preventing sealing with collodion and iodoform. Healing by second intention occurred and was very protracted, as some of the buried sutures which required removal were very deeply situated and difficult to find, but

eventually the wound closed nicely, and the bitch became quite normal in appearance. I also had a couple of cases of inguinal hernia recently in which the omentum was extensively adherent to the sac.

SARCOMA OF THE INTESTINE OF A DOG.

By Professor J. J. O'CONNOR.

Dublin.

Subject.—An aged Scotch terrier dog.

History.—Three or four weeks ago the dog passed blood copiously from the rectum, it being reckoned that a teacupful was passed at a time. Two or three fragments of bone were also passed, and it was thought that they were the cause of the bleeding, but it continued after their expulsion, the fæces, which were soft, being always mixed with blood.

Symptoms.—The dog was very thin but in fairly good spirits, and feeding well. The visible mucous membranes were extremely pale. There was no evidence of abdominal pain. On manipulation of the abdominal wall a fairly large, somewhat doughy mass could be felt but its nature could not be definitely ascertained, and there was no pain evinced on its being compressed. It could not be an obstruction in the bowel as there was no evidence as such. The condition was diagnosed as either ulceration or malignant disease of the bowel.

Treatment.—Treatment for ulceration of the bowel was tried for a few days, but as the patient became rapidly worse, getting very weak and emaciated, he was destroyed with chloroform. *Post-mortem* examination revealed a thickening of the entire circumference of the bowel for a distance of about 4 in., the wall of the bowel being about ½ in. in thickness and fibrous in appearance and consistency. Professor Mettam made a microscopic examination of the lesion and found that it was a sarcoma. There were other small thickenings in the course of the bowel, and the lining of the organ in the region of the large tumour was hæmorrhagic.

Abstracts.

ERADICATION OF TICKS BY THE STARVATION METHOD.

By H. E. LAWS, B.Sc., F.I.C., and B. MANNING.

Gonubie Park, East London, South Africa.

The part played by the different species of ticks in the transmission of various diseases amongst stock and domestic animals is unfortunately too well known to all owners of stock running on the coast veld of the Cape Colony to be cited here.

If the presence of the tick is essential to the propagation of the disease, then tick eradication will eventually result in its extermination.

We know of no case where any species of tick, and hence the disease conveyed by it, has been completely exterminated, but the ravages of many have been reduced, and the disease controlled in a number of instances. Our work here during the last few years has proved beyond doubt that it is possible for any persevering farmer to keep the ticks in a state of suppression to such an extent that losses from the diseases conveyed by them are reduced to a negligible quantity. This is emphasized particularly by the fact that, in the early stages of our experimental work, losses amongst sheep and calves due to heartwater were frequent and numerous.

Cases of heartwater amongst our calves are now practically non-existent, and during the later stages of our sheep grazing no deaths whatever occurred from this disease.

The methods hitherto adopted for eradication of ticks are:—

(1) Periodic dipping of the hosts.

(2) Grass burning.

(3) Enclosing of defined areas for a sufficient length of time to ensure all ticks dying off through the absence of hosts.

We will deal with these three methods categorically.

(1) *Dipping of Hosts.*—Dipping is undoubtedly the best method for eradicating ticks, provided an efficient dip be used. We have proved by a series of experiments that the most efficient and economical dips contain arsenic in some form, although a number of other substances are almost as effective in destroying ticks, and some of these can be used with advantage in conjunction with arsenic.

Dipping must be periodic; the intervals between each dipping varying with the species of ticks which are to be eradicated.

Cattle dipping has been in progress here for the last five years, but it is only for the past two years or more that stringent methods have been adopted. The interval between each dipping is fourteen days, and the eradication is carried out by a system of rotation. Each camp is overstocked in turn with cattle, which are dipped fortnightly, until the pasturage is so low that the animals cannot maintain their condition; then they are moved to another paddock and the work continued there until the same stage is reached. The cattle are then either moved into a third paddock or returned to the first. This overstocking reduces the natural covering for the ticks, and the cleaning of each paddock is therefore done in a minimum amount of time.

By this means we have practically now not only exterminated every species of tick, but have also improved the condition of the veld, an achievement which is impossible when burning or starvation is adopted.

(2) *Burning of Grass.*—This has been practised by a number of farmers, and doubtless burning does destroy a certain number of ticks if it is done when the ticks are on the top of the grass.

The best time to burn for the destruction of ticks is in March and April; but even then only a small proportion of the ticks are destroyed, and if no other means are adopted, total eradication must be regarded as out of the question. Burning is also to be deprecated on account of the impoverishment of the pasturage which must of necessity result.

(3) *Starvation.*—Or enclosing of defined areas for a sufficient length of time to ensure all ticks dying off through the absence of hosts. If a tick is confined in an enclosed space for a sufficient length of time it will eventually die of starvation. Experiments have been carried out by Mr. Lounsbury and others to ascertain the period which the different species of ticks will survive without access to hosts, and a few experiments in this connection have been done in the laboratory here, with the following results:—

On November 4, 1908, fully engorged female bonts, browns, blues and reds, were placed in separate tubes in the incubator. In due course the laying was completed and the larvæ hatched out.

On June 1, 1909, the larvæ of all species had started to die, the browns showing less life than the others.

On December 16, 1909, all the browns were dead, but some of the bonts, blues and reds were still alive.

On February 1, 1910, (fifteen months after), a few bonts and reds were still alive, but all the blues were dead.

By the end of March, 1910, all the bonts and reds were dead.

From these results one would conclude that if an area be enclosed so that no possible host has access to it the ticks in that area would be exterminated well within the space of two years. Theoretically this would be the easiest way of eradicating ticks, but in practice many difficulties arise which complicate matters. Not the least important of these is due to the fact that it is almost impossible to enclose a paddock of sufficient dimensions for the requirements of this experiment so that no ground game nor birds of any kind can have access to it. These will not only act as hosts for the ticks (in the paddock) which desire to feed, but will also be the means of bringing in ticks from beyond the enclosed area.

The idea of enclosing a small space free from bushes, about one or two acres in extent, by means of double fences situated about 10 ft. apart, the outside being supplied with netting to keep out all game, was first of all considered, but was discarded as being impracticable. If this course was adopted, although the grass could be burnt at the commencement in order to drive out all game, we would still have to contend with birds. Our experiment, strictly speaking, does not give us any information regarding the period ticks will survive without feeding on a host: it was merely intended to ascertain the effect of keeping pasturage free from stock for considerable periods, and so compare its influence on the extermination of ticks with overstocking

of similar pasturage, and dipping at fortnightly intervals. We desired to make a practical test which could be undertaken by any farmer who is willing to allow parts of his veld to lie idle for a time.

The camp selected at Gonubie Park for the purpose is enclosed with a single 4 ft. 6 in. six-strand wire fence, one barbed and five plain, about 160 acres in extent.

Down the centre of the camp runs a ravine, the banks of which are covered with brushwood and trees, which spread, in one part, into a bush about one hundred yards wide. The lower portion of the camp is flat pasture land with coarse ferns distributed amongst the grasses. In the upper portion the one side of the camp is covered with scattered mimosa trees, notoriously excellent covering for such ticks as bonts, which require shade. In summer the grass is often 3 ft. high in this portion. On the other side of the ravine are clumps of bushes which afford splendid cover for ticks. The grass was only of a moderate height between these bushes, being of the variety commonly called "rooi grass."

Owing to the nature of the area enclosed, game, especially hares and duiker, is fairly plentiful; and although several determined efforts were made to drive them out during the time the camp has been enclosed, yet the retired position of the camp and the absence of foreign life within its precints were sufficient inducement for the buck and hares to return to it.

Several duiker have been shot at different times in the camp, and on examination we have found them well infested with larval and nymphal reds, browns, and buck ticks (*Ixodes pilosus*).

On May 1, 1908, the camp was enclosed, and at the end of twelve and eighteen months, respectively, clean cattle were put into it for three days, when, on examination, they were found to be well infested with ticks.

On February 2, 1910, twenty-one months after the camp had been closed, its degree of infestation was again tested. Three beasts were cleaned of ticks by hand, and then sprayed with a mixture of paraffin and water to kill any larval or nymphal ticks which might be concealed by the hair. They were then driven into the camp and allowed to remain there for three days, *i.e.*, until February 5, 1910. On examination, after being taken out, the following ticks were found:—

Two red ticks (*R. evertsi*).
Forty-four Cape brown (*R. capensis*).
Two brown (*R. appendiculatus*).
Three blackpitted brown (*R. simus*).
Three male and one female bont (*Amblyomma hebraeum*).

The above gives a total of fifty-five adult ticks. The number of cattle was so small, and the area so large, that the number of ticks found does not convey any idea as to the degree of infestation of the camp. After this examination of the camp, and on account of the results obtained, we proposed to burn the grass before making our next test. We were approached by Mr. Lounsbury and Mr. Borthwick, who asked us to allow the camp to remain unburnt. Finally it was decided to burn one half and leave the other; then erect a temporary fence dividing the burnt from the unburnt portion.

The burning of the camp was done thoroughly on March 22, 1910. That portion which contains the most thorn bushes was burnt, and

the more open veld left. The erection of the fence was commenced immediately afterwards within the burnt portion, leaving a margin of about 30 ft. wide of burnt patch running right down the unburnt side of the fence, to prevent any ticks from the unburnt crawling through to the burnt section.

The Fourth Examination of the Camp.

The Burnt Section.—On May 10, 1910, ten beasts, which had been dipped on May 7 in the Gonubie Bath, were cleaned of ticks by hand, and immediately driven into the burnt portion of the Starvation Camp. They were taken out seventy-two hours after being put in, *i.e.*, on May 13, and immediately examined, with the following results:—

On the ten beasts only four adult ticks were found. Owing to such a scarcity of ticks on these ten beasts it was decided to repeat the experiment. The same ten beasts were driven back into the burnt portion of the camp on May 13. On May 17, *i.e.*, ninety-six hours later, they were taken out and immediately examined, with the following results:—

On the ten beasts thirty-two adult ticks were found. In addition, one beast was found to be infested in the bottom of its ears with larval and nymphal reds.

On the conclusion of this examination it was decided to compare the state of the burnt section of the Starvation Camp with some of the farm camps which are being cleaned in rotation by overstocking and fortnightly dipping, and have not been burnt for six years at least.

On May 17 these cattle, after thorough cleaning by hand, were put into the largest camp on the farm, at the Gonubie Mouth, and allowed to run there until May 20, when they were taken out for examination.

On the ten beasts only twenty adult ticks were found. They were also free from larvæ and nymphs.

The Unburnt Section.—At 5.30 p.m. on May 10, ten beasts were cleaned of ticks and driven into the unburnt portion of the Starvation Camp, and remained there until 9 a.m. on May 14, when they were subjected to examination. On the ten beasts twenty-one adult ticks and one nymph were found. In order to make a true comparison between the degree of infestation of the burnt and unburnt section of the camp, these ten beasts were put back into the unburnt portion of the camp and allowed to remain there until May 17, when they were brought out and examined the second time. In each instance the cattle were prevented from grazing on the burnt margin near the temporary fence. On the ten beasts thirty-seven adult ticks were found. In addition, one beast was found with larval reds in its ears.

These ten beasts were, after being cleaned, put into the "Sea Camp," another of the camps near the foreshore, on May 17, where they remained until May 20, when they were taken out and examined. On the ten beasts only sixteen adult ticks were found.

In the first part of this experiment the cattle were in the unburnt portion a few hours longer than those in the burnt portion, but even allowing for this, there are fewer ticks in the burnt section than in the unburnt. This bears out the theory that grass burning at the right season destroys a number of ticks.

Conclusions.

From the above it is obvious that the method of starvation reduces considerably the number of ticks within the enclosed area, and that, if starvation be combined with the burning of the grass, the numbers are still further reduced; but so long as game have access to the enclosed area, total eradication is out of the question, the large majority of the ticks remaining there being species which are often found on all kinds of game, particularly bush buck and duiker.

The tremendous difference in the number of ticks found at the first and second tests of both portions of the Starvation Camp is probably due to the fact that the cattle had recently been dipped at the time the first test was made, which gives the cattle a certain protection for a short time. The results of the second tests can be taken as normal; and these we must take as our standard when preparing the degree of infestation of this Starvation Paddock with the other grazing paddocks on the farm. When this standard is taken we find that both the grazing paddocks are cleaner than the burnt portion of the Starvation Paddock, and infinitely cleaner than the unburnt portion.

Thus we have proved beyond doubt that periodic dipping in an efficient dip at intervals of not more than fourteen days is a superior process for the extermination of ticks of all kinds than starvation, and at the same time the former method has the advantage over the latter in that it can be carried on in conjunction with the ordinary farm work. Incidentally the nature of the pasturage is considerably improved by the former process, whereas the latter tends to impoverish it.

Our experimental work here was undertaken primarily with the object of eradicating bont ticks—hence the fortnightly interval was adopted for each dipping.

Seeing that we found only two bonts in the two camps, covering an area of 1,000 acres, or more than one-third the extent of the farm, the results may be regarded as highly satisfactory. But there are still as many browns and reds in these camps as in the burnt portion of the Starvation Camp. This is on account of the fact that these species stay on the host for such a short time that the majority of them escape the dippings. Fortunately they require only a weak dip to destroy them, and this can be used at more frequent intervals than that which is required to kill female bonts.

It is quite possible that in due course, if our work here is continued on the same lines as in the past, we shall be as successful in exterminating brown ticks as we have been with bonts; but, in view of the fact that East Coast fever is approaching the Colony, this process would necessarily be considered far too lengthy with such a long interval between dippings on those farms where ticks have been allowed to increase, and no means have hitherto been devised for controlling them.

To meet the requirements of such cases, dipping (or spraying) every seven or five days would be necessary. This process would incidentally destroy all other species of ticks.

(*Agricultural Journal, Cape of Good Hope.*)

TECHNIQUE FOR OBTAINING OPSONIC INDICES.

By Major J. B. ANDERSON.

Royal Army Medical Corps.

There are plenty of knowledgeable persons about who are ready to explain what opsonic indices are and how to take them, and I am aware much has been written in the different periodicals on the subject, but the difficulty is where to find the matter. My object in writing these few lines is to supply this want, as when at home I searched the booksellers for literature on the technique and could find none. By chance I came across Dr. Houghton's "Review of Opsonins," and when taking a course at St. Mary's Hospital was given some excellent leaflets on the subject.

For those not instructed in the rudiments or principles of the subject, it might be well to ask the question, What is an "Opsonic Index"? The answer resolves itself into saying that a film or smear is made on an ordinary slide from a mixture (that has been incubated a little) of (1) washed blood corpuscles; (2) bacterial emulsion; (3) patient's serum. This film is appropriately stained, and when examined under the $\frac{1}{12}$ oil immersion it is seen that phagocytosis has taken place. The microbic contents of, say, 100 polymorphonuclear neutrophiles are enumerated, and the number divided by 100 will therefore give the average number of bacteria in each white cell; this is called the phagocytic count or content, and should not be less than about three per cell, except for tubercle, which should be about two per cell. A phagocytic count is made in a similar way with (1) washed blood corpuscles; (2) bacterial emulsion; (3) normal or pooled serum; and this latter divided into the former gives the "opsonic index" of the patient against the particular bacterium used in the emulsion.

An example might make it clearer.

(1) From film made with patient's serum it is found that 100 polymorphonuclear cells contain 350 bacilli or cocci.

(2) From film made with control, normal, or pooled serum, 100 polymorphonuclears contain 280 bacilli or cocci; then 3·50 will be the patient's phagocytic count, and 2·80 the control count. Therefore, $3{\cdot}50 \div 2{\cdot}80 = 1{\cdot}25$, and this is the "Opsonic Index" of the patient against the particular bacillus or coccus used in the emulsion, 1·0 being approximately the normal. The technique employed may appear at first simple, but it is only by constant practice that anything approaching reliable results can be obtained. The pitfalls are many, so it is as well at first not to be too dogmatic about conclusions until all technicalities are overcome and one's results are more or less consistent.

It will be seen that for the estimation of opsonic indices three essentials are necessary, viz.:—

(1) Sera, viz., (*a*) serum of patient whose opsonic index is required; (*b*) serum of normal person for control.[1]

(2) Washed corpuscles.

(3) Emulsion of bacteria.

[1] As one normal serum alone does not give a fair estimation of a normal index, it is usual to take two or three normal sera and mix them. This is called a "pooled serum." The index of each may also be taken separately, and the average of these considered as the normal.

These we will now proceed to prepare *seriatim*, and I place sera first, as during the preparation of the other two time is given for the serum to separate out.

(1) *Sera.*—Both the patient's and normal sera are best collected in the usual manner in glass capsules, which must not be over heated when sealing in the flame. The blood should also be fresh, as after about five days the fluids have lost about half their opsonising powers. By placing the capsules and their contents into an opsonizer, a name given to a convenient and simple form of incubator, at 37° C., for from a quarter to half an hour, the sera will separate from the clot more quickly.

After the clot is formed, and not before, the capsules may be placed in a centrifuge to accelerate the separation if necessary. If the clot has not formed, plasma instead of serum will separate out, and this, when mixed with the washed corpuscles and emulsion, will produce clotting, thus nullifying results. There is no reason why serum alone should not be sent for examination, put up in a sealed capillary tube, provided, of course, it is not too old when received.

(2) *Washed Corpuscles.*—Take a clean glass tube about 2 in. long and $\frac{1}{4}$ in. to $\frac{1}{5}$ in. in diameter, one end of which is closed. Fill two-thirds of this tube with 1·5 per cent. citrate of soda solution, and the remaining one-third with ordinary blood from the finger.

Before and after centrifuging place the tube with its contents between the forefinger and thumb, and invert it three or four times to mix the citrate solution and blood. It must not be shaken. Centrifuge till the corpuscles just settle. Too much centrifuging is apt to compress the red cells, and cause the leucocytes to adhere to each other. With a pipette and teat, remove as much as possible of the citrate solution, adding in its place 1·86 per cent. sodium chloride solution. Wash by inverting (as was done with the citrate of soda solution) for a minute or so, then pipette off the salt solution. Mix the remaining blood cells intimately by means of a pipette and teat, and a homogeneous mixture of washed corpuscles will be obtained, which will keep for twenty-four hours.

(3) *Emulsion of Bacteria.*—This is the most difficult part of the technique. It is essential that the living growth from which the culture, and afterwards the emulsion, is made be a pure one. The age of coliform and Gram-negative cocci should be from about four to ten hours, as if older, they are unlikely to take the stain. Staphylococcal growths on agar may be twelve to twenty-four hours old. Streptococcal growths on agar about the same age, but three or four tubes should be prepared, as the growth is usually scanty. Gonococcal[1] growths usually should be four to eight hours, but may be sometimes ten to twelve hours. Emulsions of tubercle bacilli, as will presently be seen, are not usually made directly from a living culture, as the process of obtaining a growth is difficult and tedious. They can be conveniently and easily obtained from dry tuberculin powder.

Having obtained a satisfactory growth, take a loopful and emulsify it with two or three drops of 0·85 per cent. NaCl solution in a clean watch-glass. Avoid pouring salt solution on to the growth in the tube. The emulsifying is more satisfactorily carried out with a mixing pipette, attached to which is a rubber teat.

[1] Best grown on agar, the surface of which is spread with sterilized serum.

By holding the pipette perpendicularly to the watch-glass the emulsion can be alternately sucked up and forced out for five or more minutes, which action breaks up any clumps. Streptococcal growths usually require more emulsifying than staphylococcal, in order to get rid of the chains. If necessary, more salt solution may be added, but the important point is to get an emulsion of the right consistency, which is a matter of practice, and achieved by educating the eye on looking through a glass stump[1] containing some of the material, and seeing how translucent it is and whether of the right milky opalescence. Invariably it has to be centrifuged, and this is best done whilst in the stump, to precipitate the clumps. Afterwards the supernatant fluid is pipetted off and placed in a similar stump, which, again, must be scrutinised with the naked eye. If it still appears too thick, more centrifuging can be done, or more salt solution added, or both. Bacillary emulsions always appear more milky and turbid than coccal ones; in the latter the opacity is but slight. Having obtained what may be considered a good emulsion, it must now be put to the test to see whether it is so or not. In order to do this equal quantities of washed corpuscles, emulsion, and serum, in the order named, are taken into a throttled pipette, separated from one another by an air space. The teat is compressed, and the contents are driven on to a clean slide. These are now thoroughly mixed with a mixing pipette, into which the mixture is finally drawn. The pipette is then sealed off and placed in the incubator or opsonizer for about fifteen minutes. The contents are then blown out on to a slide, cleaned by rubbing it gently for a few seconds with very fine emery paper, then cleaning with a duster (this method is as good as using alcohol and ether). Films are now made and appropriately stained, and examined with the $\frac{1}{12}$th. If the emulsion is a good one, *i.e.*, not too thick or too thin, each phagocyte ought to contain two or three bacteria.

An emulsion of tubercle bacilli is prepared somewhat differently, and is made from either a fresh culture or from dead tubercle germs, the latter being the more convenient method, as the germs are easily procurable in the form of a powder. Take a little of this powder—about half as much as can be got on to a threepenny piece—and put it into an agate mortar, and with a pestle thoroughly grind down: 1·5 per cent. salt solution is now added drop by drop to form a paste. This percentage is used, as with a smaller amount of salt the solution favours phagycytosis, producing what is known as spontaneous phagocytosis. It might be here mentioned that the same percentage is used for making emulsions of Gram-negative cocci, such as the gonococcus and *Micrococcus neoformans*—a coccus found at the edge of cancerous growths. The grinding is still continued, and a little more of the diluent is added till the paste becomes an emulsion. This part of the process takes from eight to ten minutes. Now place the emulsion in a special bulbed tube, when it can be further mixed for two or three minutes with a mixing pipette. The contents are then placed in a clean test-tube, and made up to about 4 c.c. with the salt solution. The test-tube is then closed in a flame, at the same time drawing out a neck. It may now be shaken in a machine called a shaker, or else

[1] A glass stump is best made by breaking a throttled pipette at its neck, then sealing off.

by hand, for about half an hour or more. Let it settle for a minute or two. Now break the neck, and take about 2 c.c. of the clean fluid and place it in a glass stump; centrifuge three or four minutes to precipitate any clumps, and pipette off the supernatant fluid, which is put into another glass stump. Its opalescence is now tested with the naked eye. If it appears too thick, it may again be centrifuged, or else some 1·5 per cent. salt solution may be added, provided there are no clumps. Presuming now an emulsion has been obtained of the right consistency, a little can be put up with washed corpuscles and serum as was done previously: then, if found necessary, it may again be centrifuged or diluted, or both.

Having described in detail the preparation of the three essentials, it now becomes necessary to describe how these are put up, then how the smear is made, stained, and, finally, to show how the counting is done.

(1) *How to put up the Test.*—A pipette, to which is attached a rubber teat, is taken, and into it are drawn equal quantities of the washed blood corpuscles, emulsion, and serum, each occupying about an inch of the tubing, and being separated from each other by an air space. These are then put out on to a slide by compressing the teat, and then intimately mixed by alternately drawing up and expelling the mixture three or four times. When finally it is drawn up for the last time, air spaces must be carefully avoided as they interfere with phagocytosis. The end is then sealed off in a flame, and the pipette placed in the opsonizer for fifteen minutes.

(2) *How to make the Smear.*—Having removed the pipette from the opsonizer, nip the end of it off, and put a small drop out on to a clean slide and make a smear. The smear is best made with a special spreader, which greatly simplifies the counting. Its spreading edge ought to be sharp and very slightly concave. It can be made thus: Take an ordinary glass slide and nick the middle of each of its long edges with a file. Take the slide between the forefinger and the thumb of each hand, and bend it sufficiently till it snaps across. After a few attempts a half of one of the slides will be found to possess a satisfactory shape and slightly concave edge. Nick off the corners of this edge with the assistance of a file, so that the smear when made will not occupy the whole breadth of the slide.

The smear should be equal and regular, and contain nearly all the leucocytes at its extremity. The idea of the concavity is to pass over the red cells, yet not to pass over the white cells, which it draws to the end of the smear.

(3) *How to Stain the Specimen.*—Films may be stained with Leishman in the ordinary way, or with carbol-thionin ($\frac{1}{4}$ per cent. thionin, 1 per cent. carbolic acid) for quarter of an hour. Tubercle films should be fixed with formalin and not with perchloride, as the white cells get broken up when stained with the carbol-fuchsin. Place the film over the vapour of formalin for five seconds, then pour on boiling carbol-fuchsin and leave for ten minutes. Wash in tap-water, and then place in 2 per cent. H_2SO_4 till the colour just rises, which will be in a few seconds, wash, then add 4 per cent. acetic acid for a moment, washing it off almost immediately. The acetic acid washes out the red cells, which would otherwise overstain with methylene blue, which is now added as a counter-stain, for a minute or so, when it is washed

off and the slide dried. This methylene blue has a strength of ½ per cent., to which ½ per cent. sodium carbonate is added.

(4) *How to Count.*—This having been touched upon at the beginning of the subject there remains but little to add. Should the number of bacteria be counted in, say, only fifty polynuclears, then the result must naturally be multiplied by 2 before dividing by 100 to get the phagocytic count, which is necessary, as I have already shown that the opsonic index is the phagocytic count of the patient divided by the phagocytic count of the pooled serum. I think the best method of counting is to enumerate the number of bacteria in five leucocytes at a time. An example will render this clear.

Number of bacteria in 100 leucocytes in series of five at a time. Patient:—

16	32	30	11	20	25	2	23	17	18	=	194
5	33	12	44	19	30	14	21	12	19	=	209
								Total	...		403

Number of bacteria in 100 leucocytes in series of five at a time. Pooled serum:—

25	19	20	12	8	13	11	18	22	8	=	156
7	27	13	23	30	12	11	12	22	5	=	162
								Total	...		318

Therefore $403 \div 100 = 4{\cdot}03$ phagocytic count of patient; and
$318 \div 100 = 3{\cdot}18$,, ,, pooled sera.

Therefore $\frac{4{\cdot}03}{3{\cdot}18} = 1{\cdot}26$ = opsonic index.

(*Journal of the Royal Army Medical Corps.*)

Reviews.

MANUAL OF TROPICAL MEDICINE. By A. Castellani, M.D., Member of the Royal Society's Commission on Sleeping Sickness in Uganda, and A. J. Chalmers, M.D., F.R.C.S., D.P.H., Colombo. Demy 8vo., pp. xxx. + 1242, with 373 illustrations and 14 coloured plates. Published by Messrs. Baillière, Tindall and Cox, London. Price 21s. net.

Drs. Castellani and Chalmers are to be congratulated in having produced in the manual a work of great excellence, and one which is really indispensable to all practitioners and students of Tropical medicine. The authors have had almost unique opportunities of becoming directly and intimately acquainted with the subject matter of the volume, which, of course, renders it much more valuable than it could otherwise be. The results of the latest investigations are all included, and the whole volume is well up to date. The work consists of three parts. Part I. occupies 84 pages and is introductory, including a history of tropical medicine, tropical climatology, and an account of the incidence of disease in the Tropics, referring especially to the occurrence of the various diseases in

different parts of the Tropics and the different races attacked. Part II. occupies about 500 pages and deals with the causation of disease in the Tropics, both physical, chemical, and biological. The morphology of the various biological causes is excellently described; in fact this section is probably more complete than any standard work on Tropical parasitology.

Part III. deals with the diseases of the Tropics, and completes the work. Each disease is described systematically under the headings Synonyms, Definitions, History, Climatology, Etiology, Pathology, Symptomatology, Diagnosis, Prognosis, Treatment, and Prophylaxis. In some cases it has been considered necessary, and we think rightly so, to repeat in some degree the description of parasites given in Part II. This procedure renders the context more readily understood.

The numerous illustrations, which are very clear and well produced, will be found of the greatest service to students.

The work is one which should be in the hands of all practitioners and student of tropical medicine and all investigators of tropical diseases.

The manual, which is one of the University series, is well up to the usual excellence of production of the publishers.

THE ENCYCLOPÆDIA OF SPORT. Parts III., IV., and V. Profusely illustrated with drawings, photographs, and coloured plates. Complete in about thirty parts, issued fortnightly. Published by Mr. W. Heinemann, London. 1s. net.

We have already appreciatively referred to Parts I. and II. of this work, and can now speak in similar terms of Parts III., IV., and V. Part III. concludes the excellent article on automobiles, and also deals with badger-hunting, badminton, baseball, and bear-shooting. It commences an excellent article on big game, which is continued in Part IV., and occupies about fifty most excellently illustrated pages. Billiards is the next subject taken in hand, and so much is to be said on the popular game and its variations—English and French billiards, snooker, pool, pyramids—that over forty pages are required, the subject being carried over to Part V. This part contains well-written articles on bird's-nesting, bison and buffalo hunting, and wild boar shooting, bowls, boxing, and bull-fighting. One point we regret to notice in the later parts is a falling off in the number of coloured plates.

Translations.

A CASE OF CEREBRAL TUMOUR.

By M. A. PICARD.

Veterinary Surgeon to the 21st Dragoons (France).

A MARE of the third squadron was brought to the infirmary, and presented very pronounced cerebral trouble. She bored at the wall, had very violent crises, during which she fell about on the objects surrounding her. At the end of twenty-four hours she died; a diagnosis of meningo-encephalitis had been given.

The lesions found at the autopsy were very interesting, it having been made chiefly on the nerve centres. The dura mater was congested, and showed in places some thickenings like milk-white plates; the pia mater and all the encephalic portion were congested, especially at the level of the inferior face of the bulb where the dilated vessels formed a dark red arborization. The left hemisphere, on simple inspection, seemed more congested than the right. The whole of the encephalus was œdematous, and of rather abnormal volume. A horizontal section of the two hemispheres passing through the upper face of the corpus callosum was made. On arriving at the right lateral ventricle a considerable quantity of citron yellow liquid was discharged. The left lateral ventricle was occupied by a yellowish, fibrinous mass, and at first sight very vascular on its surface. This mass was 6 c.m. long, and extended into all the frontal cornu from the ventricular crossway; this mass, more voluminous than the ventricle, was folded on its walls, and compressed the oval centre, which was slightly congested. The anterior extremity of the frontal cornu extended to within 1 c.m. of the frontal extremity of the left hemisphere, and was on a more anterior plane than that of the right ventricle.

This tumour was carefully dissected out and enucleated. We noticed that it was adherent by its posterior part to the upper choroidean tissue, which seemed to form a pedicle; the choroid plexuses were prolonged on the upper edge of the tumour up to its anterior part. This yellowish tumour resisted the impression of the finger and allowed serum to transude in large quantity; cut longitudinally a resistant and reddish tissue was encountered. Preserved in a solution of formalin, and examined after hardening, the neoplastic tissue had become white, and on section we encountered a whitish tissue studded with little black islets.

Histological examination by Professor Curtiss, of Lille, revealed a hard, round-celled sarcoma, with large spaces of mucous tissue dominating all the preparation, and coloured a uniform red with picro-carmine. Evidently we had to deal with a myxo-sarcoma originating in the dura mater.

(*Revue Générale de Médecine Vétérinaire.*)

HÆMOPNEUMOTHORAX IN THE DOG DUE TO THE PENETRATION OF AN EAR OF CORN INTO THE LUNGS.

By Professor J. POENARU.

Hæmopneumothorax represents a discharge of sanguinolent liquid and air into the thoracic cavity. This affection, which happens very rarely in our domestic animals as a complication of tuberculosis, gangrene, cancer, pulmono-pleural hæmatoma, is still rarer when it is due to a foreign body such as an ear of wheat, which, penetrating from the stomach into the thoracic cavity, causes lesions in the pleural serosa and the lung, and sets up traumatic hæmopneumothorax.

The subject of our observation is a French setter, strong and muscular, with which his master went out sporting in the month of August, 1909. The following day he observed that his dog was dull, that it ate very little, remained lying about, vomited often, and was weak, This state lasted until the month of October, when the animal commenced to cough and expectorate blood.

At the time of examination the animal presented the following symptoms: dulness; respiratory muscles atrophied; eyes sunk in the orbit; ocular and buccal mucosæ pallid; labial breathing; sanguinolent discharge; respiration dyspnœic and very superficial; a pronounced orthopnœa; intercostal spaces prominent and convex; pulse feeble and accelerated, temperature 38° C. On auscultation, bronchial râles and a particular inspiration resembling an amphoric râle; vesicular murmur diminished on account of a clear sound exaggerated by percussion in the superior parts of the thoracic cavity; in the inferior region a dull sound of metallic character. The painful cough was accompanied by an effort at vomiting, which threw off by the nose and the mouth a sanguinolent and foamy liquid—a veritable hæmoptysis.

From similar symptoms previously seen, the diagnosis of tuberculosis with pneumothorax was given. The tuberculin test gave negative results, however. The symptom of hæmoptysis was treated with injections of gelatinized serum, with five drops of adrenalin and 1 milligramme of ergotinin Tanret in subcutaneous injections. However, the illness progressed, and the animal died on October 30.

Autopsy.—Six hundred grammes of blood in the thoracic cavity and the pulmonary lobes atelectesied on their inferior borders; the diaphragmatic face of the pulmonary lobes adherent to the diaphragm; a pleuro-pulmonary diaphragmatic symphysis (dry pleurisy).

The superior edge of the right pulmonary lobe traversed from above to below and from behind to in front by an ear of wheat, causing a pulmonary rupture covered with clots of blood.

On the anterior face of the diaphragm a pimpled wound, which corresponds on its opposed face to an adherence of the stomach, in its depth the wound is completely cicatrized. Around the pulmonary wound one observes a hæmorrhagic congestion, and in the bronchi and trachea frothy blood.

As a *résumé*, the evolution of the malady must have been the following: The dog in his track across the field swallowed a whole

ear of wheat; by movement and efforts provoked by vomiting this ear became implanted on the wall of the stomach and penetrated it and the diaphragm, causing a fistulous wound, which produced first an adherence between the stomach and diaphragm, followed by dry diaphragmatic pleurisy with pleuro-pulmonary symphysis.

The pulmonary lobes being fixed by a tissue of new formation to the diaphragm, their posterior face has become more bound down, and in this way the ear of wheat has passed through the pulmonary lobe from below to above, producing a wound large enough to cause hæmothorax at the same time as pneumothorax.

(*Arrhiva Veterinaria.*)

AN OBSTETRICAL OBSERVATION.

By MARTIN.

A cow, about eight months pregnant, had been straining for three days, and ejecting a brown liquid of bad odour. The general state was good. Manual exploration showed a posterior presentation, and that the envelopes were putrefied. Parturition was effected without much difficulty, a normal calf being produced in an apparent state of death; after ten minutes' artificial respiration it was resuscitated. Afterwards its development was quite normal. The mother died some hours after calving.

The autopsy showed metritis and septic peritonitis (30 litres of brown chocolate exudate in the peritoneum).

This observation shows that a calf can continue to live in a violently inflamed womb surrounded by putrefied envelopes.

(*Münchener Tierärztliche Wochenschrift.*)

A CASE OF ŒSOPHAGISMUS.

By BERTON.

A MARE, before being operated on, received 20 grm. of sulphonal in 100 grm. of dry bran. During the whole operation she struggled. After getting up she was led into the box, and took some mouthfuls of hay from the rack. But suddenly mastication was arrested, the physiognomy became anxious, the legs stiffened, the head was extended on the neck and suddenly stretched out and shaken with spasms which extended over the whole body. These spasms were accompanied by audible groans, and followed after a few seconds by a bilateral nasal discharge of copious viscous material, greyish-white, of salivary appearance; and this in its turn was followed by an inodorous, liquid, greenish vomit containing particles of chewed hay.

During three-quarters of an hour these phenomena were renewed ten times, with progressively decreasing intensity. The material thrown up became less and less abundant, and more and more clear, until finally it was purely salivary. Some hours afterwards only prostration remained; there was no other bad sign. It was only three days afterwards that a myositis of the cervical region became evident.

(*Revue Vétérinaire Militaire.*)

PYOMETRA OF THE BITCH.

By KARL KELLER.

INFECTION results *post partum* after an abortion, or at the time of œstrum, in fact as long as the orific. extern. is open to a certain degree. Very seldom infections occur in animals which have not been lined or borne young. For pyometra to occur there must be endometritis and a collection of secretion in the uterus. The formation of the secretion occurs through injury to the internal muscular tissue of the uterus which becomes inactive, and unable to contract and open the mouth of the womb. The cause of the affection is a streptococcus, colourable by Gram; besides one finds in the pus of pyometra a bacillus with specific morphological peculiarities.

On section one finds the horns of the uterus thickened to the size of a child's arm, and the wall of the uterus 1 to 5 mm. thick. Often the widenings of the uterine horns are ampulla like. The purulent material is brown-red, brown-violet, or occasionally hæmorrhagic or yellowish-green, of thin, slimy to doughy consistency. Many a time the uterus contains stinking gases. Microscopically one finds the superficial epithelium club-shaped or inflated, the glands widened, soaked with slime or pus, the muscle bundles disintegrated by infiltrations and the filaments atrophied.

Clinical symptoms are vaginal discharge, increase of the belly circumference, slight intermittent fever, emaciation, forward vaulting of the belly, inertness, stiff gait, failure of appetite, thirst, polyuria, and as a complication, purulent cystitis and albuminuria.

Diagnosis is made from the vaginal discharge and by local examination, the absence of ichorous tumours may be determined. By palpation one can make out the fluctuating mass.

Differential diagnosis comprises the making out of pregnancy and ascites. Prognosis bad, if not treated early.

Therapy.—Total extirpation alone cures old, weak animals; those with subnormal temperature, heart deficiency, and cachectic appearances are inoperable.

(*Zeitschrift für Tiermedizin.*)

ON THE TREATMENT OF STOMACHAL INDIGESTION OF THE HORSE BY MAREK'S METHOD.

By NIEDER.

PROFESSOR MAREK, in cases of alimentary surfeit or gaseous dilatation, systematically practices sounding of the stomach, with the view of provoking forced regurgitation and relief of the organ by evacuation of its contents. He invented a special sound, and gave precise directions for its use, which Behrens later on simplified. M. Nieder adopted the technique described by the latter. He recommended "holding the animal's head down and letting the tongue go free," and used the cuneiform gag of Bayer.

The sound introduced into the mouth, the passage of the pharyngeal isthmus into the œsophagus constitutes the most delicate part of

the operation; it always provokes a certain amount of agitation, and sometimes the anguish of suffocation. These disquieting symptoms, of short duration, are deprived of ensuing harm if the operator "has some *sang froid* and a light hand." The penetration of the sound into the trachea is recognized by the great ease with which it passes, and and its sudden arrest at the bronchial bifurcation; the column of air expired, which escapes by the extremity of the sound, confirms the error of situation. In such a case the apparatus is immediately withdrawn, and a fresh attempt made. A short initiation is sufficient to surmount all difficulties. No external sign indicates the moment of penetration into the stomach.

Marek and Behrens advise taking note of the length of the sound introduced, and consider it has reached its destination when it has passed in for about 2 metres on horses standing about 1·60 metres in height. The mandarin withdrawn after a more or less prolonged waiting, according to circumstances, there is produced, in the cases of gaseous or liquid surfeit, an evacuation of gas or discharge of a liquid like dregs of wine, with a sourish odour. In surfeit, properly so called, a very small quantity of gas only is evacuated. With the help of a funnel adapted to the free extremity of the sound, some litres of warm water are introduced into the stomach, and regurgitation takes place thirty-five or forty minutes afterwards. The operation is completed by a veritable lavage of the stomach by means of 5 or 6 litres of hot water. After rubbing down and a rectal douche, the invalid is left to himself, and the cure is complete in less than an hour. One sounding alone is generally sufficient.

Out of six reported observations five cures were obtained. The one failure should not be imputed to the method, for there was a rupture of the stomach before the sounding of the organ.

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Books and Periodicals, &c., Received.

Proceedings of the Royal Society of Medicine; Journal of the Royal Army Medical Corps; Bulletins of the Bureau of Sleeping Sickness; Agricultural Journal of the Cape of Good Hope; Transvaal Agricultural Journal.

Letters and Communications, &c.

Professor O'Connor; Mr. E. Ryan; Mr. J. L. Perry; Mr. J. Varney; Board of Agriculture and Fisheries; Department for Agriculture and Technical Education for Ireland; Bureau of Animal Industry.

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